ASIAN JOURNAL OF PHARMACEUTICAL AND CLINICAL RESEARCH

INNOVARE ACADEMIC SCIENCES
Knowledge to Innovation

Vol 10, Issue 6, 2017

Online - 2455-3891
Print - 0974-2441

Case Report

A CASE REPORT ON CHRONIC LEAD POISONING FROM OCCUPATIONAL EXPOSURE

NAZMA M, DHIVYA K*, GNANA CHAITANYA C, DIVYA SREE P, LAKSHMI PRASANNA S

Department of Pharmacy Practice, Sri Venkateswara College of Pharmacy, Chittoor - 517 127, Andhra Pradesh, India.
Email: divyapharmd@gmail.com

Received: 21 February 2017, Revised* and *Accepted: 30 April 2017

ABSTRACT

The hazard to public health from lead continues to be a matter of concern. It is one of the most serious environmental poisons among the toxic heavy metals all over the world. Lead poisoning is seen in all age groups, especially in adults working in lead-based industries. We report the case of a 28-year-old man working in an unorganized lead-based manufacturing unit admitted with the complaints of giddiness, excessive tiredness, pain in the upper abdomen, decreased appetite, excessive body pains, increased sweating, and oliguria. Investigations carried out during the admission showed hemoglobin levels of 8.5 g/dl and blood lead level (BLL) of 115 µg/dl. The patient was subjected to chelation therapy. After repeated course of chelation therapy, he has shown the signs of improvement. The paucity of a safe workplace and awareness among workers results in high BLLs. Therefore, education and awareness related to lead hazards is considered necessary.

Keywords: Lead poisoning, Kidney damage, Chelating agents, Industrial workers.

 DOI: http://dx.doi.org/10.22159/ajpcr.2017.v10i6.18061

INTRODUCTION

Lead is a pervasive metal with unique properties such as softness, high malleability, ductility, low melting point, and resistance to corrosion which has been used by mankind for many years [1]. The hazard to public health from lead continues to be a matter of concern. It is one of the most serious environmental poisons among the toxic heavy metals all over the world. People are using lead for many years because of its wide variety of applications as building materials, pigments, pipes and in the manufacture of kitchen utensils, trays, and other decorative articleHuman exposure to lead is from innumerable sources, and the major sources are contaminated air, food, dust, soil, and water; use of certain products containing lead such as lead-soldered cans, traditional practices such as herbal or folk remedies, lead bullets, jewelry, cosmetics, artisan ceramics, environmental emissions containing lead production; through occupations such as motor vehicle assembling, lead smelting, refining, alloying and casting, battery manufacturing and recycling, lead paint, jewelry, welding and pottery [4,5]. Lead poisoning is seen in all age groups, especially in adults working in lead-based industries are benighted of the ill effects of lead. The mainstay of treatment for lead toxicity is chelation therapy with oral succimer, penicillamine or parenteral dimercaprol edetate, and calcium disodium edetate [6]. Here, we report a case of 28-year-old man who was diagnosed with chronic lead poisoning.

CASE REPORT

A 28-year-old male admitted to the hospital with complaints of giddiness, excessive tiredness, pain in the upper abdomen, decreased appetite, excessive body pains, increased sweating, and oliguria. He is a smoker, occasional alcoholic, married, and no significant allergic history was found. Occupational history revealed that he had been working in an unorganized lead-based manufacturing unit for 4 years. On examination, his pulse rate was 82/minutes and blood pressure was 124/60 mmHg. Results of various laboratory investigations were normal except hemoglobin - 8.5 g/dl (normal range: 14-16 g/dl), blood urea-62 mg/dl (15-45 mg/dl), serum creatinine - 1.7 mg/dl (0.6-1.2 mg/dl), zinc protoporphyrin (ZPP) - 126 µg/dl (up to 40 µg/dl), and blood lead level (BLL) - 115 µg/dl (acceptable range 10 µg/dl). Peripheral smear revealed microcytic hypochromic anemia. On the basis of these findings, the diagnosis of chronic lead poisoning was made. The patient was advised to change his occupation and treated with three courses of chelation therapy using the chelator D-penicillamine (25-35 mg/kg/days) in divided doses along with supplemental measures. After repeated course of chelation therapy, he showed some signs of improvement. He responded with an improvement in hemoglobin to 12.8 g/dl. The blood level of lead and ZPP before and after chelation therapy was shown in Table 1.

DICUSSION

In the present study, the patient was unaware of the ill effects of lead and was handling lead without taking any precautions even though they have provided protective equipment, resulted in chronic lead poisoning. Elevation of serum creatinine and blood urea level indicated kidney damage as a result of chronic lead exposure. Several studies explained various effects on the body based on serum lead level which includes teratogenicity when the maternal BLLs are at a concentration of 10-15 µg/dl, reduced prostate and seminal function at BLLs of 40-50 µg/dl, neurological symptoms, anemia at a concentration of 40-70 and 50-80 µg/dl, respectively, brain and renal damage at serum concentration of 100 µg/dl in adults and 80 µg/dl in pediatrics. Death due to lead poisoning is seen at serum concentration of 125 µg/dl in pediatrics [7,8]. Lead in the body comprises 2% in the blood (t1/2=35 days) and 95% in bone and dentine (t1/2=20-30 years). Blood lead may remain elevated for years after cessation from long exposure, due to redistribution from bone. Blood lead concentration is the most widely used marker for inorganic lead exposure. ZPP concentration in blood usefully reflects lead exposure over the prior 3 months [9,10]. The toxic effects of lead are majorly due to higher affinity to thiol- and phosphate-containing ligands thereby inhibiting biosynthesis of heme and affecting membrane permeability of major organs such as kidney, liver, and brain resulting in altered functioning and degradation of these tissues. Lead also acts by inhibiting aminolaevulinic acid dehydratase thereby blocking the conversion of aminolaevulinic acid to porphobilinogen which leads to elevated levels of aminolaevulinic acid results in decreased gamma-aminobutyric acid release [11]. The clinical manifestations of lead poisoning: Nervous system include headache, fatigue, ataxia, myalgia, seizures, hallucinations, loss of memory, mental retardation, encephalopathic syndrome; gastrointestinal system are nausea, vomiting, constipation, and metallic taste; reproductive system

Table 1: BLL and ZPP levels before and after chelation therapy

Parameters	Before therapy	After 1st course of therapy	After 2nd course of therapy	1 month after discontinuing therapy	After 3rd course of therapy
BLL (µg/dl)	126	65	52	61	45
ZPP (µg/dl)	115	59	48	63	55

ZPP: Zinc protoporphyrin, BLL: Blood lead level

include abnormal spermatogenesis, sterility in males, and miscarriage in pregnant women; hematopoietic system are anemia, elevation in protoporphyrin is usually observed; renal effects include glycosuria, proteinuria, nephropathy whereas systemic and renal hypertension is commonly observed cardiovascular manifestations. Other acute toxicity manifestations include limb, joint pain, and deposition of lead in teeth and bones on chronic exposure [12-14]. Although chelation therapy removes lead from the blood and soft tissues, on discontinuation of treatment, it is redistributed from the bony compartment to the blood [15]. This evidently suggests that the chronic lead exposure requires repeated courses of treatment. The patients undergoing chelation therapy require intensive monitoring throughout the treatment period and BLLs should be estimated at the end of each course, and the subsequent therapy should be based on this determination.

CONCLUSION

The paucity of a safe workplace and awareness among workers results in high BLLs. Therefore, education and awareness related to lead hazards is considered necessary. The regulatory bodies should make it mandatory to evaluate and create awareness among public as well as workers in lead-based industries about the ill effects of lead and should insist on regular health checkup to prevent adverse health effects. Furthermore, pharmacy professionals should assist in providing patient education, psychological support, and conducting campaign against lead hazards.

ACKNOWLEDGMENT

The authors are thankful to the management of Sri Venkateswara College of Pharmacy and RVS Institute of Medical Sciences for providing excellent research guidance and support.

REFERENCES

1. Carocci A, Catalano A, Lauria G, Sinicropi MS, Genchi G. Lead toxicity, antioxidant defense and environment. Rev Environ Contam Toxicol 2016;238:45-67.
2. Flora G, Gupta D, Tiwari A. Toxicity of lead: A review with recent updates. Interdiscip Toxicol 2012;5(2):47-58.
3. Gupta S, Manoj KR, Meena SD. Effect of selected heavy metals (lead and zinc) on seedling growth of soybean glycine max (L.) Merr. Int J Pharm Pharm Sci 2016;8(8):302-5.
4. Amol SK, Amit RN, Anurag GP, Anita S, Vinod G. Lead poisoning: An overlooked diagnosis in clinical practice. Int J Res Ayurveda Pharm 2012;3(5):639-44.
5. Papanikolaou NC, Hatzidaki EG, Belivanis S, Tzanakakis GN, Tsatsakis AM. Lead toxicity update. A brief review. Med Sci Monit 2005;11(10):RA329-36.
6. Woolf AD, Goldman R, Bellinger DC. Update on the clinical management of childhood lead poisoning. Pediatr Clin North Am 2007;54(2):271-94, viii.
7. Rastogi SK. Renal effects of environmental and occupational lead exposure. Indian J Occup Environ Med 2008;12(3):103-6.
8. Prashant A, Anitgya M, Kumar M, Tripathi SK. A comprehensive review of environmental exposure of lead and its toxicity. J Indian Forensic Med 2009;31(3):299-303.
9. Gordon JN, Taylor A, Bennett PN. Lead poisoning: Case studies. Br J Clin Pharmacol 2002;53(5):451-8.
10. Grandjean P, Jørgensen PJ, Viskum S. Temporal and interindividual variation in erythrocyte zinc-protoporphyrin in lead exposed workers. Br J Ind Med 1991;48(4):254-7.
11. Bergdahl IA, Grubb A, Schütz A, Desnick RJ, Wetmur JG, Sassa S, *et al*. Lead binding to delta-aminolevulinic acid dehydratase (ALAD) in human erythrocytes. Pharmacol Toxicol 1997;81(4):153-8.
12. Kimberlie A, Graeme MD, Charles VP. Heavy metal toxicity, part II: Lead and metal fume fever. J Emerg Med 1998;16(2):171-7.
13. Goldberg A. Lead poisoning and haem biosynthesis. Br J Haematol 1972;23(5):521-4.
14. Thenmozhi M, Dhanalakshmi M, Devi KM, Sushila K, Thenmozhi S. Evaluation of hepatoprotective activity of *Leucas aspera* hydroalcoholic leaf extract during exposure to lead acetate in male albino wistar rats. Asian J Pharm Clin Res 2013;6:78-81.
15. Schütz A, Skerfving S, Christoffersson JO, Tell I. Chelatable lead versus lead in human trabecular and compact bone. Sci Total Environ 1987;61:201-9.