

Ten-year Follow-up of Blood Lead Levels with Medical Removal Protection of Shipyard Workers

Tsan YANG¹, Ho-Jui TUNG², Jiann-Chian SHYR³, Ching-Huang LAI¹,
Ching-Hui LOH⁴ and Saou-Hsing LIOU^{1,5*}

¹ School of Public Health, National Defense Medical Center, 161 Minchuan East Road, Sec. 6, Nei-Hu, Taipei, Taiwan, 114 ROC

² Department of Humanities and Social Sciences, National Defense Medical Center, Nei-Hu, Taipei, Taiwan, ROC

³ Division of Cardiology, Department of Internal Medicine, Yee-Zen General Hospital, Taoyuan, Taiwan, ROC

⁴ Department of Family Medicine and Internal Medicine, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan, ROC

⁵ Division of Environmental Health and Occupational Medicine, National Health Research Institutes, Kaoshiung, Taiwan, ROC

Received May 6, 2004 and accepted April 25, 2005

Abstract: This cases report compared the short-term changes of BLL with medical removal intervention and follow-up the long-term changes of BLL afterward. During a physical examination in October 1992, a 44-year old shipyard welder was discovered to have a blood lead level (BLL) of 54.1 $\mu\text{g}/\text{dl}$. It was recommended that the shipyard remove this worker from his workplace. In 1993 the BLLs checked for this worker were 36.7 $\mu\text{g}/\text{dl}$ in March and 32.0 $\mu\text{g}/\text{dl}$ in April. After six months of medical removal, he returned to initial welding work. In 2002, we collected two blood samples from this worker for analysis in May and October. The results were 30.4 $\mu\text{g}/\text{dl}$ and 31.6 $\mu\text{g}/\text{dl}$, respectively. Meanwhile, two other welding workers (case 2 and case 3) with BLLs over 40 $\mu\text{g}/\text{dl}$ in the survey conducted at the same shipyard in 1992. It took 4 yr to let BLLs downed to less than 40 $\mu\text{g}/\text{dl}$. However, after the blood lead concentration drops to below 40 $\mu\text{g}/\text{dl}$, 10 yr long-term observation indicates that BLLs reduction level off and do not continue to go down in these three cases.

Key words: Shipyard, Welder, Blood lead level, Medical removal protection

The risk of lead exposure in shipyards is an old problem¹⁻⁴. Many studies showed that workers involved in welding work are especially prone to increased blood lead concentrations⁵⁻⁷. In the past, only few contributions to the long-term change of blood lead levels after the medical removal have been reported. A study described the fall in zinc protoporphyrin levels (ZPP) in patients with chronic lead intoxication. The ZPP levels fell rapidly from a high level to almost normal levels within six months⁸. Another case report indicated that blood lead concentration fell dramatically from 240 to 42 $\mu\text{g}/\text{dl}$ after the last known lead exposure⁹. In Taiwan, because of advances in technology and improvements in

environmental conditions, few cases of shipyard workers with blood lead level higher than 50 $\mu\text{g}/\text{dl}$ have occurred since the 1990's. Although medical removal is recommended to be one of the control measures for lead poisoning workers, the implementation of medical removal is somewhat difficult in Taiwan. For the employers, they are reluctant to remove productive and experienced workers. At the same time, workers may not report their minor symptom that may be related to lead exposure in order to keep their job, unless it is a lead-poison case. As a result, it is rare for physicians in Taiwan to directly observe the transition of BLL before and after a medical removal. The ten years follow-up of BLLs in shipyard workers without chelating agent intervention was even missing in Taiwan. This cases report compared

*To whom correspondence should be addressed.

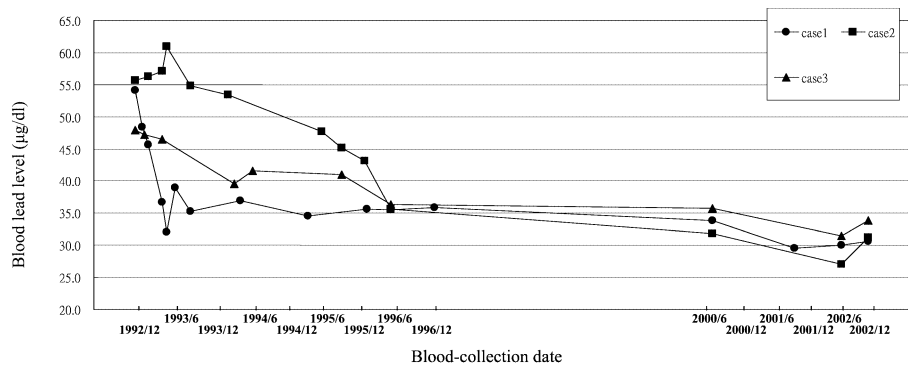


Fig. 1. Long-term observation of blood lead levels in three shipyard welders.

the short-term changes of BLL with medical removal intervention and follow-up the long-term changes of BLL afterward.

The BLLs were measured using a graphite furnace atomic absorption spectrophotometer (GFAAS, GBC 3000 system). All instruments were soaked in a 20% nitric acid solution for 48 hours in order to remove potential lead contamination. Certified whole blood controls were selected for the accuracy test (Nycomed Pharma Co. Oslo, Norway). To avoid the blood specimen “matrix effect” during analysis, the standard addition method was used to build up the calibration curve. The detection limit for the analysis was 0.3 µg/dl. To ensure the quality of BLL testing, our laboratory has a consistent intra-laboratory quality control and has participated in the blood lead proficiency test program of the United States CDC (Centers for Disease Control and Prevention) since 1992.

This study presents ten-year follow-up of a shipyard welder with an original blood lead level of 54.1 µg/dl. He was 44-year old, when he was identified in 1992, and had been engaged in welding since 31 years old. His main job was to repair the body of the ship. Welding and cutting on workpieces that have metallic coatings may be especially hazardous. Lead-based paints have been used commonly on marine and structural membrane. Welding on these surfaces during repair and shipbreaking generates high lead fume concentrations^{2, 10, 11}. Physical examination indicated that, except for the unacceptable blood lead level, the worker was normal in other physiological and diagnostic tests. The worker’s personal information revealed that he was a non-smoker and he did not drink either. He also denied any lead-related disease history. He wore the appropriate work clothes, put on his anti-dust mask, drank milk occasionally, and washed his work clothes every day. Overall, this worker’s personal health habits were considered good. Aside from

the present on-the-job lead exposure, there were no other plausible lead contamination sources. Consequently, we concluded that the increase in the worker’s blood lead level was caused by lead exposure on the job.

Acting on our recommendations, the factory immediately transferred the worker from his job to one involving document handling. The worker (case 1) was, however, very concerned about his blood lead level. He went to hospitals for some blood lead tests each year. His BLLs spread and observation time are recorded in details in Fig. 1. Case 1 showed a significant reduction in the worker’s blood lead level. From the beginning of the medical removal to six months after, there was a significant reduction in the worker’s blood lead level (from 54.1 µg/dl to 31.6 µg/dl).

Meanwhile, two other welding workers (case 2 and case 3) with BLLs over 40 µg/dl in the survey conducted at the same shipyard in 1992. Their data are plotted and added to Fig. 1. These two workers did not receive medical removal, so their blood lead concentration did not change dramatically as what was seen in case 1 and it took 4 yr for their BLLs to drop to less than 40 µg/dl. This finding is consistent with a previous report regarding medical removal¹². Therefore, we speculate that medical removal may be an effective measure to avoid chronic lead poisoning.

Case 1 went back to his original job at the end of his medical removal. However, after the ending of medical removal, BLL in case 1 showed a little rebound on April of 1993 and then showed gradually decrease. During this period, the shipyard asked industrial hygienists to implement hazard control measures and had gradually improved their ventilation system. Figure 1 shows the natural progression of these workers’ BLLs. Because of improvements in ventilatory technology, the BLL of case 1 decreased to around 35 µg/dl since the 1994’s and then downed to 30 µg/dl after 2001. The BLLs of those two cases without medical removal were

also decreased to around 35 $\mu\text{g}/\text{dl}$ since the 1996's and then were also downed to around 30 $\mu\text{g}/\text{dl}$ after 2001.

The distribution of lead in the bloodstream is largely concentrated in the bones and the half-life of the lead in the bone is about 15 to 20 yr. As a result, the lead level in bone can be used as an index for long-term lead exposure. The blood lead level does not account for the total accumulated volume in the body, but rather represents the lead exposure for the past two months^{13, 14}. Consequently, it is possible that lead concentration changes could be caused by another form of lead exposure outside the body or lead emissions from the bones. This is especially true when the body suffered infection, fever, pregnancy, or hyperthyroidism, etc.^{15, 16}. Currently, it is believed that many nutrients such as calcium, iron, zinc, phosphorus and foods such as milk, vegetables and tofu can help control the increase of lead in the blood^{17–20}. However, from our personal inquiries, this worker reported that his diet habits remained unchanged through the period. From Fig. 1, we can see that the BLL in case 1 went down to below 40 $\mu\text{g}/\text{dl}$ about six months after the medical removal. Similar changing tendency was observed in several previous studies^{8, 9, 12}. For instance, O'Flaherty (1986) compared the length of time under three different medical removal mandates (80/60, 70/50, and 60/40 $\mu\text{g}/\text{dl}$). They reported that, under a 60/40 $\mu\text{g}/\text{dl}$ removal rule, the median length was between 6 to 8 months among the employees with over 60 $\mu\text{g}/\text{dl}$ BLLs could be allowed to work again¹². During this medical removal stage, the reduction in blood lead level was more rapid, which may have something to do with the lifespan of the red blood cells (RBC's). However, the BLL of case 1 stabilized after 1994, when he went back to his original work environment. It should be noted that the shipyard's gradually improving ventilation system and requiring its workers to use personal protection devices all play a major role in reducing the average BLLs. The efficacy of hazard control can be demonstrated by a significant reduction of BLLs among the same shipyard employees over a 10-year period. According to the data from follow-up surveys, the average BLLs of the 158 workers had gradually decreased from 23.4 ± 10.5 $\mu\text{g}/\text{dl}$ (ranging from 4.2 to 57.2 $\mu\text{g}/\text{dl}$) in 1992 to 10.4 ± 5.9 $\mu\text{g}/\text{dl}$ (153 workers, ranging from 3.2 to 34.8 $\mu\text{g}/\text{dl}$) by 2002. More importantly, not a single welding worker had blood lead concentration higher than 40 $\mu\text{g}/\text{dl}$ in 2002 (data not shown). This study discovered that when a long-time (23 yr since first exposure) shipyard worker (case 1) is transferred away from a high exposure environment, there is a significant decline in BLL. However, when the BLL reaches around 30–35 $\mu\text{g}/\text{dl}$, it stabilizes and stops declining without further interventions. During a

medical removal period, the rate of decline of BLL should be influenced by the magnitude of return of lead from peripheral tissues to the blood. The amounts of lead stored in peripheral tissues reflect both magnitude of exposure and, especially for slowly exchanging tissues such as those that account for much of the body burden of lead in adult¹², during of exposure, they reflect integrated total exposure, or duration \times magnitude.

In our cases, the BLLs did not drop to normal range (10 $\mu\text{g}/\text{dl}$ or below). A plausible explanation could be that the half-life of lead in bone needs 15–20 yr, which was longer than only 10 yr of observation in this study. Furthermore, the workers resumed their original work, so that they were still being exposed to few lead fumes even though the factory environment has been improved. Under the situation when lead accumulates inside the body for a long time, the observation over a 10-yr period indicated that it is not easy for the BLL to reach the general population range of below 10 $\mu\text{g}/\text{dl}$. Both the occupational health officials and workers should be informed that a medical removal may not make BLLs return to a normal range of 10 $\mu\text{g}/\text{dl}$ (or less). According to our observations, even though the case 1 had undergone the medical removal and the factory had improved ventilation facilities, his BLL has always been higher than above 10 $\mu\text{g}/\text{dl}$ over the 10-yr observation period.

References

- 1) Rieke FF (1969) Lead intoxication in shipbuilding and shipscrapping 1941 to 1968. *Arch Environ Health* **19**, 521–39.
- 2) Tola S, Karskela V (1976) Occupational lead exposure in Finland. V. Shipyards and shipbreaking. *Scand J Work Environ Health* **2**, 31–6.
- 3) Grandjean P, Kon SH (1981) Lead exposure of welders and bystanders in a ship repair yard. *Am J Ind Med* **2**, 65–70.
- 4) Booher LE (1988) Lead exposure in a ship overhaul facility during paint removal. *Am Ind Hyg Assoc J* **49**, 121–7.
- 5) Chiang HC, Chang PY (1989) Lead intoxication in shipscrapping employees in Taiwan. *Kaohsiung J Med Sci* **5**, 284–90.
- 6) Brigham CR, Landrigan PJ (1985) Safety and health in boatbuilding and repair. *Am J Ind Med* **8**, 169–82.
- 7) Jakubowski H, Trzcinka-Ochocka M, Razniewska G, Frydrych J (1998) Blood lead levels in industrial workers in Poland. *International J Occupational Medicine & Environmental Health* **11**, 59–68.

- 8) Hryhorczuk DO, Hogan MM, Mallin K, Hessel SM, Orris P (1985) The fall of zinc protoporphyrin levels in workers treated for chronic lead intoxication. *J Occup Med* **27**, 816–20.
- 9) Williams MK (1984) Biological tests of lead absorption following a brief massive exposure. *J Occup Med* **26**, 532–3.
- 10) Landrigan PJ, Straub WE (1985) Occupational lead exposure aboard a tall ship. *Am J Ind Med* **8**, 233–9.
- 11) Rastogi SK, Gupta BN, Husain T, Mathar N, Srivastava S (1991) Spirometric abnormalities among welders. *Environ Res* **56**, 15–24.
- 12) O'flaherty EJ (1986) The rate of decline of blood lead in lead industry workers during medical removal: the effect of job tenure. *Fund Appl Toxicol* **6**, 372–80.
- 13) Rabinowitz MB (1991) Toxicokinetics of bone lead. *Environ Health Perspect* **91**, 33–7.
- 14) Nilsson U, Attewell R, Christoffersson JO, Schutz A, Ahlgren L, Skerfving S, Mattsson S (1991) Kinetics of lead in bone and blood after end of occupational exposure. *Pharmacol Toxicol* **68**, 477–84.
- 15) Silbergeld EK, Schwartz J, Mahaffey K (1988) Lead and osteoporosis: mobilization of lead from bone in postmenopausal women. *Environ Res* **47**, 79–94.
- 16) Gold RH, White R, Kales SN, Hu H (1994) Lead poisoning from mobilization of bone stores during thyrotoxicosis. *Am J Ind Med* **25**, 417–24.
- 17) Mahaffey KR (1981) Nutritional factors in lead poisoning. *Nutr Rev* **39**, 353–62.
- 18) Mahaffey KR, Michaelson IA (1980) The interaction between lead and nutrition. In: *Low level lead exposure: the clinical implications of current research*. ed. by Needleman HL, 159–200. New York Raven Press, New York.
- 19) Chen C, Wang X, Chen D, Li G, Ronnenberg A, Watanabe H, Wang X, Ryan L, Christiani DC, Xu X (2001) Tofu consumption and blood lead levels in young Chinese adults. *Am J Epidemiol* **153**, 1206–12.
- 20) Barton JC, Conrad ME, Nuby S, Harrison L (1978) Effects of iron on the absorption and retention of lead. *J Lab Clin Med* **92**, 536–47.