Lead Exposure in Migrant Children in Northern Colorado

Edward Hendrikson, PA-C, PhD
Ft. Lupton, Colorado

Lead is the most common toxic substance found in the United States, and children are most affected by lead poisoning. The numerous adverse effects occur in the hematologic, immunologic, and neurologic areas. The neurologic sequela, which are difficult to recognize, include mental and developmental delays that start early in life. One study of children of migrant farmworkers found that 24% of the children between the ages of 6 months and 5 years had increased levels of lead in their blood. A total of 21 % of the migrant farmworker housing sites had increased lead levels in their water supply. Health professionals should screen all children under the age of 5 years, especially children of migrant farmworkers. (J AM ACAD PHYS ASSIST 1994;7:707-10.)

Lead is the most commonly found toxic agent in the United States, and the potential for exposure in children is high.[1-4] People are exposed to lead through the skin, through the respiratory system, and by ingestion, with ingestion the most common way children are affected.[5] Lead poisoning is most prevalent in children aged 1 to 6 years who live in older dwellings[6]; the interior walls of older homes have lead-based paint that chips, and children ingest the particles. Children also increase their exposure by ingesting dust and soil that have increased lead levels. For example, if a child who weighs 10 kg ingests 0.41 gm of contaminated street dust, clinical lead poisoning or blood lead levels of at least 20 μg could result.[7] The concentration of lead in the air has been decreasing because of the use of unleaded gasolines; however, industrial pollution still accounts for some inhalation exposures in urban areas.[8] In addition, children who live near industrial sites increase their exposure in the summer because they tend to play outside for longer periods of time. Children who live in urban areas are at greater risk than those who live in rural areas, and the potential for exposure in children of migrant workers is probably even greater because of their living conditions.

Lead is typically a cumulative poison, primarily because it is excreted slowly. Sudden poisonings are rare but can occur. Such poisonings are usually found among adults who work in industrial settings that have high concentrations of lead.

> **Lead poisoning is most prevalent in children aged 1 to 6 years who live in older dwellings.**

Lead exposure has been found to increase the host susceptibility to infectious agents such as *Escherichia coli* and staphylococcus bacteria.[9] Children with elevated serum lead levels who were infected with shigellae have had prolonged diarrhea, possibly indicating a decreased host response to other infectious agents.[10] In laboratory animals prolonged exposure to lead caused a decrease in their IgM response.[11]

The neurologic effects of lead poisoning cause the most concern (Table 1). Lead has been shown to cause segmental degeneration of the nerve. In addition, demyelination takes place, which causes a slowdown in conduction.[12] Lead exposure during late gestational development and the early postnatal period can decrease synoptic counts in the cerebral cortex.[13-16] It

Edward Hendrikson is a PA at the Plan de Salud del Valle in Ft. Lupton, Colo.

Reprint requests: Edward Hendrikson, 1250 W. 152 Ave., Broomfield, CO 80020.

0893-7400/94/$3.00 + 0 26/1/59036

Journal of the American Academy
of Physician Assistants
November 1994

TABLE 1. BLOOD LEAD LEVELS

Level	Result
10-40 μg/dl	Decreased enzyme in red blood cell production, anemia, decreased IQ, decreased mental development, decreased IgM response
40-50 μg/dl	Peripheral neuropathy
50-60 μg/dl	Minimal brain dysfunction, convulsions
>60 μg/dl	Encephalopathy

has also been shown to inhibit the calcium-mediated release of the neurotransmitter acetylcholine, causing a decrease in synoptic action.[17]

Chelation therapy has substantially reduced the mortality rate, but at least 50% of the survivors of lead-induced encephalopathy have severe brain damage, and an even greater percentage may have learning disabilities. The prognosis is also poor after recurrent low-dose exposure to lead. This situation emphasizes the need to test children before the symptomatic stages; early detection helps prevent permanent neurologic sequelae.[18-20]

■ SYMPTOMS AND TREATMENT

Clinical manifestations include colic, which typically is poorly localized, with abdominal rigidity, weakness, irritability, weight loss, personality changes, ataxia, constipation, headaches, neuritis, confusion, and encephalopathy, which can lead to coma. The classic sign of wrist drop is less common in children, and a lead line observed on the gingival margins is rarely seen but should be checked.[21] Typically, no fever occurs, and pain medications have little effect on the abdominal pain.

Lead exposure should be considered in the differential diagnosis of all children with anemia.[18] The anemia is caused by the brittleness of the red blood cell and a defect in cell maturation. The level of erythrocyte protoporphyrin typically is elevated when the anemia is because of lead exposure. In addition, low serum ferritin and low transferrin saturation will be observed.[6] The complete blood count of children with long-term exposure to lead will often show basophilic stippling of the red blood cell, which is absent in sudden, massive ingestion of lead. A 24-hour urine lead level analysis will show increasing lead levels, but the single most effective diagnostic tool is the serum lead level. Cerebrospinal fluids show moderately to markedly elevated protein and a white blood cell count of less than 100 cells/ml. However, lumbar punctures must be performed cautiously because of the increased risk of brain stem herniating caused by the encephalopathy.[21]

Treatment for higher levels includes supportive measures with a combination therapy of dimercaprol given intramuscularly and calcium disodium edathamil given intravenously. Excessive fluid administration must be avoided. Treatment should be considered if blood lead levels exceed 24 μg/dl.[6]

Notably, the Centers for Disease Control has recently lowered the acceptable standard for serum lead levels in children. The standard went from 20 μg/dl to 10 μg/dl because of recent studies that suggested that a decrease in IQ and developmental index scores occur in children with serum lead levels of between 10 and 20 μg/dl.[20]

■ STUDY POPULATION

One important aspect of treatment that is often overlooked is the evaluation of the environmental situation and the removal of the contaminated sources. In addition, because children are the most affected by lead contamination, it is important to study the effects on children who live in areas with a high probability of exposure, such as places where migrant workers live. Therefore I tested the serum blood levels of children who had two high-risk factors with the first factor assumed (they all belonged to the migrant population). The second factor was chosen from among the following: (1) living in an industrial area; (2) living with another sibling or individual with a high level of lead in the blood; (3) living in a building that was more than 25 years old; or (4) anemia.

Lead exposure should be considered in the differential diagnosis of all children with anemia.

Some difficulties exist when attempting to study such a population, primarily because of the large numbers and the problems related to gathering data on a highly transient population. The Department of Agriculture estimates that more than 1.5 million migrants work in the United States with more than 45,000 in Colorado alone. In this study population an estimated 8000 to 10,000 migrants can be found dur-

Journal of the American Academy
of Physician Assistants
Volume 7, Number 10

ing the peak season (May to October). However, current estimates come from studies that have too many confounding factors to be accurate. For example, it is difficult to make a distinction between who is really a migrant and who is living in the community and working in agriculture. Similarly, because of the constant change in migrants' living situations, one family or group of migrants may be counted twice because of a sudden move by that group. Some of the newest research suggests looking at the number of migrant jobs and connecting that number with the number of migrants in each region. Although this may give us a better picture, studies are limited. Another problem with studies of this unique population is finding a randomized representative group to examine. The attrition rate from a randomized group of migrant farmworkers is so great that significance is hard to obtain. Because of these difficulties, this study used a chosen population and followed a descriptive pattern to help find the possibility of exposure of lead in the children of migrant farmworkers.

The CDC has lowered the acceptable standard for serum lead levels in children from 20 μg/dl to 10 μg/dl.

The first part of the descriptive study was to sample the water in all migrant camps and homes to evaluate possible contamination. A total of 100 sites were sampled, and findings were reported in milligrams per liter. Collection of water from migrant housing was conducted before the migrant season in March 1993 and during the migrant season in June 1993.

Each child had blood drawn. The samples were sent to a regional laboratory; blood lead levels were expressed in micrograms per deciliter. For the purpose of this study, water levels of above 0.015 mg/L were considered abnormal, and blood lead levels of above 10 μg/dl were considered abnormal. Both blood and water samples were evaluated through atomic absorption methods.

The third aspect of this descriptive study was to evaluate cord blood or measure prenatal serum lead levels. This evaluation not only provided two tests but also helped detect high levels of lead in children at an earlier point and thus make more of a difference.

RESULTS

The results of the water testing showed that among migrant housing sites, 21% of the locations tested positive for increased levels, with the EPA standard of 0.015 mg/L used. In addition, with the exception of two locations, all towns with high concentrations of migrant workers had average lead levels at or above EPA limits (Table 2). Urban and rural site averages were almost identical at 0.0172 mg/L and 0.0176 mg/L, respectively.

TABLE 2. LEAD LEVELS AT MIGRANT HOUSING SITES

City	Average lead levels (mg/L)
Greeley	0.029
Longmont	0.018
Ft. Lupton	0.031
Hudson	0.009
Brighton	0.015
Commerce City	0.014

The results of blood testing of the migrant farmworker children with the appropriate risk levels showed that 4 of 17 tested high for lead (24%). The range of abnormal levels was 10.2 μg/dl to 12.0 μg/dl. All four children and their parents were notified and advised to use bottled water. Two of the children's blood lead levels fell below 10 μg/dl after 2 months of this intervention only. The other two children were lost to follow-up.

DISCUSSION

The limitation of this descriptive study was the number of children tested, which perhaps resulted from the nature of the migrant working conditions and the fact that two risk levels were required for testing. An additional problem was the attempt to obtain the cord blood lead levels at the delivery of children. Because of logistic problems, only two cord blood lead levels were tested. The interesting aspect was that one of the two results was an increased level of 10.2 μg/dl. The results given suggest that lead is still a serious environmental hazard in the United States and even worse in the migrant farmworker population.

Perhaps one of the most difficult aspects of low-level exposure of lead in children is the identification of children with symptoms. The most common sign or symptom is a mild developmental delay, which can be difficult to identify. In fact, an IQ of 110 would not be considered abnormal, but this child may have had an IQ of 120 without the elevated serum lead level.

Journal of the American Academy of Physician Assistants November 1994

For this reason it would make more sense to screen the pediatric population on a regular basis.

In conclusion, as the demographic trends of the migrant population shift from rural to urban dwellings, so too are the risk factors for lead exposure increased. An additional factor is that ground water contamination in the United States has worsened during the last few decades. Considering the low cost of the screening for lead exposure, the low risk of complications from the screening procedure, and the high rate of morbidity of lead exposure and poisoning, more consideration should be given to introducing lead screening to the migrant clinic situation. ■

REFERENCES

1. Goyer RA, Rhyne B. Pathological effects of lead. Int Rev Exp Pathol 1973;12:1-77.
2. McElvaire MD, Deungria EG, Matte TD, Copley CG, Binder S. Prevalence of radiographic evidence of paint chip ingestion among children with moderate to severe lead poisoning, St Louis, Missouri, 1989 through 1990. Pediatrics 1992;89:740-2.
3. Ibrahim MA. Childhood lead poisoning: a disease for history texts [Editorial]. Am J Public Health 1991;81:685-7.
4. Zylhe JW. Preventive medicine's latest goal: getting lead out to protect children. JAMA 1991;266:315-6.
5. Norback CT, Norback JC, eds. Hazardous chemicals on file™. New York: Facts on File Publications, 1988:L001.
6. Centers for Disease Control. Preventing lead poisoning in young children: a statement by the Centers for Disease Control. Atlanta: Centers for Disease Control, 1991.
7. Purdom WP. Occupational health. In: Purdom WP, ed. Environmental health. New York: Academic Press, 1980:585.
8. Occupational Safety and Health Administration. Occupational exposure to lead: final standard. Federal Register 1978:43:52932-3014.
9. Gainer JH. Effects of heavy metals and of deficiency of zinc. Am J Vet Res 1977;38:869-73.
10. Sacks HK, McCaughran DA, Krall V, Rozenfield IH, Youngsmith N. Lead poisoning without encephalopathy. Am J Dis Child 1979;133:789-90.
11. Luster MI, Laith RE, Moore JA. Effects of lead on immune response in rodents. Environ Health Perspect 1978;23:227-32.
12. Lampert PW, Schochet SS. Degeneration of the cerebellar white matter. J Neuropathol Exp Neurol 1968;27:210-20.
13. Bull RJ, McCauley PT, Tylor DH, Crofton KM. The effects of lead on developing central nervous system of the rat. Neurotoxicology 1983;4:1-18.
14. Petrone LR. Development of infants with iron deficiency [Letter]. N Engl J Med 1992;328:575.
15. Shannon MW, Graef JW. Lead intoxication in infancy. Pediatrics 1992;89:87-90.
16. Satin SK, Neutra RR, Guirguis G, Flessel P. Umbilical cord blood lead levels in California. Arch Environ Health 1991; 46:167-73.
17. Manalis RS, Cooper GP. Presynaptic and postsynaptic effects of lead at the frog neuromuscular junction. Nature 1973; 213:254-5.
18. Vaughan VC, McKay RJ, Nelson VE. Textbook of pediatrics. 10th ed. Philadelphia: W.B. Saunders Co., 1979:473-510.
19. Needleman HL, Gannoe C, Leviton A, et al. Deficits in psychologic and classroom preference of children with elevated dentine lead levels. N Engl J Med 1979;300:689-95.
20. Needleman HL, Gatsonic CA. Low-level lead exposure and the IQ of children: a meta-analysis of modern studies. JAMA 1990;263:673-8.
21. Kemp HC, Silver HK, O'Brian D, eds. Current pediatric diagnosis and treatment. 6th ed. Los Altos, California: Lange Medical Publication, 1980.