

Virtual Mentor

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Clinical Pearl

Diagnosing Pediatric Lead Toxicity

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An extraordinarily abundant though toxic element, lead has proven useful to humanity since ancient times and continues to be a key component of many products and industries. Cosmetics, food flavoring, fiber optics, pottery, batteries, paints, radiation shields, glass, and, of course, plumbing (the Latin for lead is *plumbum*) are just some of the many historical and present-day examples of the diverse applications of this metal. However practical, the “widespread dissemination of lead in the human environment” negatively impacts our health and well-being as the ubiquitous substance is (often unknowingly) ingested, inhaled, or absorbed transcutaneously [1]. Acute and chronic lead poisoning, sometimes called plumbism, are associated with significant medical morbidities, adversely affecting the renal, hepatic, hematologic, gastrointestinal, and neurologic systems. Unfortunately, the pediatric population is particularly susceptible to the neurological damage caused by acute and chronic lead poisoning [2].

One of the more alarming findings of the National Health and Nutrition Examination Survey II (1976-1980) was evidence that more than 85 percent of American preschoolers had elevated levels of lead in their blood [1]. Significant regulatory actions have since succeeded in decreasing the incidence of elevated blood lead levels in the US population. The overall prevalence of elevated blood levels (defined as >10 µg/dL) for all ages was 0.7 percent in 1999-2002 [3]. The decline in lead poisoning during the last 3 decades has been attributed to the passage and enforcement of federal legislation that effectively eliminated lead from its 3 major environmental sources: gasoline, food and beverage cans, and paint [4]. However, lead prevention must remain a public health priority. Some 24 million homes in the United States reportedly “still contain substantial lead paint hazards, with 1.2 million of these units occupied by low-income families with small children” [5]. This and other data from surveys conducted in 2000 by the Centers for Disease Control and Prevention (CDC) suggest that primary care and emergency physicians who work with children, particularly the disadvantaged, must continue to be vigilant about screening for and assisting families affected by lead poisoning [4].

National Recommendations for Pediatric Lead Screening

Two large, national medical institutions, the Centers for Disease Control and Prevention and the American Academy of Pediatrics (AAP) have developed comprehensive guidelines for physicians and health care workers engaged in pediatric lead screening [4, 6]. Physicians should also refer to state- or locale- specific screening recommendations. These targeted recommendations may be based on epidemiologic data (often organized by zip code using geographic information systems and models)

that are more relevant to the patient population immediately served. Also, the CDC web site provides links to state-specific strategic elimination and local childhood lead poisoning prevention programs. Physicians hoping to be effective advocates for lead prevention in their communities may find it useful to review these plans.

Recommendations encouraging the universal screening of all toddlers (prevalent during the 1990s) have given way to calls for more targeted screening of at-risk children based on such criteria as residence in a high-risk neighborhood or red-flag responses to personal-risk questionnaires. Currently both the CDC and the AAP advise screening all Medicaid-eligible children as well as children who are enrolled in other assistance programs like WIC (women, infants, children).

In March 2000, a 2-year-old girl living in a New Hampshire apartment complex constructed before 1920 became the first victim of fatal pediatric lead encephalopathy in over 10 years. She died 3 weeks after emigrating from Egypt with her Sudanese refugee family. According to the CDC report, “a wall in a sibling’s bedroom had multiple holes from which the patient had been seen removing and ingesting plaster” [7]. This case calls attention to the special risk that children who are refugees, adoptees, or recent immigrants face in terms of lead exposure (related primarily to poor housing conditions here in the US). Venous blood tests taken from 96 immigrant children at 90 days and then again at 3-6 months after arrival in the US demonstrated a dramatic increase (40 percent) in elevated blood lead levels [8]. The CDC therefore recommends that all refugee children between the ages of 6 months and 16 years be screened both at the time of arrival and then 3-6 months after placement in a permanent housing situation [8].

Signs and Symptoms

Mild lead poisoning (10 to 25 $\mu\text{g}/\text{dL}$) caused by repeated exposure over a period of time can be insidious. There may not be any obvious or specific physical signs or symptoms. One pediatric text advises that “plumbism should be included in the differential diagnosis of anemia; seizure disorders; severe behavioral disorders; mental retardation; colicky abdominal pain; and the arthralgia, bone pain, and cerebral and abdominal crises of sickle cell disease” [9]. In confirmed cases, lead poisoning must be taken seriously. Though the mechanism of its toxicity is not yet known, even mild exposure is capable of causing great developmental and psychological harm including cognitive impairment with lower IQ scores, impulsiveness, difficulty with concentration and attention, irritability, hearing loss, and speech delays. Indeed, a recent study identified a 7-point IQ loss in association with the first, initial 10 $\mu\text{g}/\text{dL}$ of elevated blood lead concentration during the lifetime of the patients studied [10]. This new data suggests that exposure at levels (<10 $\mu\text{g}/\text{dL}$) currently considered “safe” may in fact be dangerous with possible permanent neurologic consequences.

With more significant lead exposure (>40 $\mu\text{g}/\text{dL}$), a child may experience abdominal pain, anorexia, constipation, headaches, emesis, confusion, muscle weakness, seizures, alopecia, and anemia (classically with basophilic stippling).

At levels $>70 \mu\text{g}/\text{dL}$, there should be emergent concern about nephropathy and encephalopathy with increased intracranial pressure, impaired consciousness, bradycardia, hypertension, papilledema, respiratory depression, and coma [3].

Labs and Other Tests

Suspicion of lead poisoning is confirmed by measurement of the blood lead levels using venous samples. Hemoglobin, hematocrit, and iron studies may be ordered for evaluation of iron deficiency and anemia, conditions that are often associated with lead poisoning. An abdominal radiograph is done when there is concern about ingestion of larger lead-contaminated materials. Finally, follow-up blood monitoring is a critical, though often neglected, component of ongoing treatment and prevention [11].

It is also important to do a detailed environmental, nutritional, and developmental assessment for children with elevated blood lead levels. The goal of the environmental assessment is to identify the sources of lead exposure (eg, lead paint, lead in water, lead in imported goods, lead related to caregiver's activities) [see Table 1].

Table 1: Key Questions to Ask Regarding Lead Exposure in a Child's Environmental History

I. Paint and Soil Exposure

- Age and general condition of the primary residence and other relevant sites
- Duration of child's habitation at residence and other relevant sites
- Evidence of chewed or peeling paint on woodwork, furniture, or toys
- Recent repairs or renovations
- Outdoor soil exposure (soil contamination)

II. Relevant Behavioral Characteristics of the Child

- Hand-to-mouth activity
- Pica (unusual appetites, eg, for clay, dried paint)
- Hand washing before meals and snacks

III. Exposures to and Behaviors of Household Members

- Occupations and hobbies of adult household members

IV. Miscellaneous Questions

- Access to imported foods, cosmetics or folk remedies
- Food storage in imported pottery or metal vessels
- Presence of vinyl mini-blinds manufactured overseas before 1997
- Well water usage

Source: Centers for Disease Control and Prevention, Atlanta, GA. [12]

Prevention and Treatment

The first goal of lead poisoning treatment is to identify and then avoid or remove

(when possible) the source of lead exposure. Residential investigation and testing may include evaluating samples of house dust, paints, tap water, and bare soil. Caregiver education about the sources of lead and the neurodevelopmental hazards of lead exposure is critical to prevention [2]. Toys, pacifiers, and hands should be washed frequently. Unfortunately, the relative efficacies of most environmental lead removal techniques are less than ideal [13]. Specialized cleaning methods like high-efficiency particulate air (HEPA) vacuuming and interior dust abatement must be done frequently in order to be effective at reducing lead levels [14]. Residential paint hazard remediation is efficacious when pre-abatement blood levels are greater than 35 µg/dL [13]. This intervention entails either removing the lead paint (by such methods as sanding, heat stripping, or wire brushing) or covering the lead paint with a new surface or a binding material. Permanent removal and decontamination of environmental sources of lead can be time-consuming, costly, and inconvenient and may even require temporary relocation while the work is being done.

The National Advisory Committee on Childhood Lead Poisoning Prevention has developed an evidence-based series of recommendations for managing elevated blood lead levels [12]. According to this monograph, chelation therapy should commence at blood lead levels >45 µg/dL. Oral succimer may be used, or, if the patient is hospitalized, calcium disodium edentate (calcium EDTA) can be delivered intravenously.

At extremely high blood lead levels (>70 µg/dL) or in children with symptoms of serious lead poisoning, the appropriate treatment is parenteral therapy with EDTA and hospitalization [4]. Another agent, dimercaprol (or BAL) forms a nonpolar compound with lead that is excreted in bile and urine [2]. Because dimercaprol is water-soluble and therefore readily crosses the blood-brain-barrier, it may be particularly useful in treating acute lead encephalopathy (in conjunction with EDTA).

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