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Taking the Lead With Lead Poisoning

A child whose blood lead level is elevated for the third time in a year is brought to the ED. This case study reviews common sources of lead exposure, signs of lead poisoning, and guidelines for management in the pediatric population.

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Case

A 2-year-old boy presents to the ED after his mother is informed that her son's venous blood lead level (BLL), which was obtained 3 days prior, is 59 µg/dL (normal, <10). Further history provided in the ED includes two previous episodes of elevated BLLs in the past year that required outpatient treatment with succimer. During these past two episodes, it was suspected that the child may have been exposed to lead from paint chips, although evaluation of the home was unable to identify a source. The patient's last treatment was approximately 4 to 5 months earlier and he has since undergone periodic testing for lead. His mother denies that the child has any symptoms consistent with chronic lead poisoning, such as constipation or abdominal pain, and feels the child has both normal behavior and development.

Physical examination reveals a well-developed child with the following vital signs: blood pressure, 90/50 mm Hg; pulse, 100 beats/min; respiratory rate, 15 breaths/min; and temperature, 98.9°F. Findings on cardiac, pulmonary, and abdominal examinations are normal. Neurologic examination demonstrates normal attentiveness, motor function, and developmental milestones. The patient's initial blood analysis reveals a hemoglobin level of 8.1 g/dL and a mean corpuscular volume of 50 fL (normal, 80 to 100 fL); a peripheral blood smear is negative for red blood cell basophilic stippling. (Note that the patient's blood was obtained by venipuncture, not from a capillary blood source. Dermal contamination may lead to unreliable results with capillary testing, but low results are considered acceptable.) Urinalysis results are normal. Radiographic studies are negative for radio-opaque foreign bodies within the abdomen but positive for dense metaphyseal bands at the distal radius.

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What are important sources of lead exposure to consider in children?

In addition to identifying and managing patients who present with lead poisoning (whether acute or chronic), the clinician must also attempt to locate the source of lead exposure. Unless the child is removed from exposure to the source, he or she is at continued risk. It is possible that other family members, such as siblings, may also be exposed. Sources of lead exposure can generally be classified as environmental, occupational, and miscellaneous (such as ethnic-related foods or supplements). Lead-based paints (typically containing the white pigment lead carbonate) still remain an important source of lead poisoning in children, especially in homes built before 1978.[1,2] The Table lists common sources of lead.[3]

Table. Environmental Lead Sources

Source	
Leaded paint	Indoors, particularly in homes built before 1978 (still used outdoors)
Dust	From deteriorated lead paint
Soil	From areas tainted with deteriorated lead paint/industrial lead or leaded gasoline emissions from previous era (banned in US since 1976; still in use worldwide)
Water	Leached from lead-containing plumbing (pipes, solder), cooking utensils, water coolers
Air	Industrial emissions
Food	Lead solder in cans (pre-1991 US; still found in some imported canned foods), "natural" calcium supplements, "moonshine" whisky, lead-foil-covered wine bottles, contaminated flour, paprika

Adapted from Henretig.[3]

What are the signs and symptoms of lead poisoning in children?

The signs and symptoms of lead poisoning are diverse and depend on both the dose and duration of exposure.

The most dramatic presentation of acute pediatric plumbism (clinical lead poisoning) is lead encephalopathy. It is characterized by pernicious vomiting, altered mental status, seizures due to cerebral edema, and increased intracranial pressure. Physical exam findings may reveal oculomotor palsy, papilledema, and other signs of increased intracranial pressure. Acute lead encephalopathy occurs most often in children ages 15 to 30 months and often is associated with a BLL of greater than 100 µg/dL.[4]

Subencephalopathic symptomatic plumbism is associated with BLLs greater than 70 µg/dL, but it can occur at BLLs as low as 50 µg/dL. Signs and symptoms can range from chronic hyperactive behavior to developmental delay. Less common features can include seizures and peripheral neuropathy.[5]

The largest group of children with chronic lead poisoning comprises those with elevated BLL without any overt symptoms. The primary concerns with children in this group are the subclinical effects of chronic, low-level lead exposure. Subtle effects can include disturbances in growth, hearing, fine motor movement, and neurocognitive development.[6]

What are the critical actions to consider in the management of lead poisoning in children?

The treatment of lead poisoning in children may require an aggressive approach. For most patients, the critical action is the removal of the child from the source of the lead exposure, which in many cases may be the child's home. If the child is asymptomatic, placement in the home of family or a friend will avert admission to the hospital. Children with large gastrointestinal burdens of lead paint chips may benefit from prompt initiation of whole-bowel irrigation to reduce absorption.

Therapy with chelators, considered a mainstay of therapy for symptomatic patients, involves a complicated risk-benefit decision for asymptomatic children with an elevated BLL. Chelation therapy increases lead excretion, reduces BLLs, and allows reversal of hematologic markers of toxicity during therapy. The use of combination chelation in the treatment of acute lead encephalopathy in the 1960s contributed to a large decline in mortality. The role of chelation in subclinically symptomatic patients with mild to moderate lead burdens is less clear, with lingering questions regarding both efficacy and safety.[7]

A large multicenter trial evaluated the effect of chelation therapy with succimer on neuropsychological test scores in children with no overt symptoms but moderately elevated BLLs (20 to 44 µg/dL).[8] All children in the study were removed from the source of lead exposure and were randomized to receive either succimer or placebo. Children who received succimer initially had a rapid and significant decrease in BLLs, but a rebound was observed within 1 week, likely due to mobilization of lead from bone. The placebo group also had a decrease in BLLs, probably due to removal from the exposure. At the end of the study period, the two groups had similar BLLs, and no differential improvement in cognition, behavior, or neuropsychological function was demonstrated. Given the lack of clear

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benefit and the cost of treatment, the findings from this study suggest that the use of chelation in asymptomatic patients should be undertaken only after careful consideration. However, it should be noted that determining that a young child is truly asymptomatic is fraught with difficulty, as the baseline level of function is limited, complicating assessment of subtle changes.[8]

What is the recommended chelation protocol for children?

The decision to use chelation therapy for childhood lead poisoning, as well as the selection of a chelator, depends on many factors, such as age, BLL, and clinical findings. Decision making should involve consultation with a medical toxicologist, a department of health, or a pediatrician with experience in management of lead poisoning. Three chelating agents are currently recommended for the treatment of lead poisoning. BAL (British anti-Lewisite), also called dimercaprol, is an intramuscular drug that is formulated in peanut oil due to its insolubility in water; it is associated with painful injection. Edetate calcium disodium ($CaNa_2EDTA$) is administered intravenously, either alone or in combination with BAL, for symptomatic patients and asymptomatic patients with extreme elevations in BLL. Succimer, or dimercaptosuccinic acid (DMSA), is available for oral therapy and is used in patients with mild to moderate poisoning.[9] Treatment guidelines from the New York City Department of Health and Mental Hygiene (www.nyc.gov/html/doh/downloads/pdf/lead/lead-chelation.pdf)[10] and the American Academy of Pediatrics[7] are also valuable resources.

The following steps should be taken prior to initiation of chelation therapy in children with a BLL of 45 µg/d[10]: (1) a venous blood specimen should be taken and processed emergently (unless encephalopathy is apparent) to confirm that the child's BLL is 45 µg/dL or greater; (2) abdominal radiography should be performed to rule out ingestion of solid lead; if radio-opaque particles are detected or if the child was seen ingesting lead, a cathartic should be given; (3) admission and chelation therapy should be arranged in a hospital with experience in the treatment of children with lead poisoning; (4) discharge to a lead-safe environment should be ensured; the local department of health should be informed of hospitalization for lead poisoning and should inspect the child's home prior to discharge.

Case Conclusion

The child received oral succimer and was admitted to the hospital for further evaluation. On the third day of succimer therapy, results of a repeat specimen taken on admission revealed a BLL of 71 µg/dL. At this time, the intravenous chelating agent $CaNa_2EDTA$ was added. The repeat BLL, drawn on the third hospital day, with results known the following day (4 days after initiation of this therapy), was 21 µg/dL, prompting discontinuation of the $CaNa_2EDTA$. Succimer was continued for the full 21-day course. Further investigation by the department of health confirmed that the home was not the source of lead, and questioning for other sources, such as home remedies, was inconclusive. Results of testing in one sibling were unremarkable. **EM**

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