

LEAD: AN OVERVIEW FOR HEALTHCARE PROFESSIONALS

Lead is a naturally occurring heavy metal. Due to its extensive use in many areas of industry and its prolonged half-life, its presence in the environment is now ubiquitous. It has many health consequences to pediatric patients.

Sources of Exposure

Many of the major sources of environmental lead exposure have been greatly reduced. Two initiatives in the 1970's lead to a dramatic decrease in blood lead levels: the virtual elimination of lead in gasoline and prohibiting the use lead-based paint in residential homes. Lead-based paint is still a problem in older homes painted before this legislation. Besides chipping paint, the soil around older houses could be contaminated by particulate lead. Other sources that remain a problem today include: industrial sites, glazed pottery, alternative medicines, and ingested lead foreign bodies.

Routes of Exposure

1. Gastrointestinal - Ingestion of lead in the form of dust, paint chips, or foreign bodies is the major form of absorption for children. Furthermore, children have a much higher rate of gastrointestinal absorption than adults.
2. Inhalational - With the elimination of lead from gasoline, this mode of exposure is chiefly in adults in an occupational setting (like smelting).
3. Dermal - Not a significant form of exposure in most cases.

Key Pharmacokinetics Points

When lead is first absorbed it enters the bloodstream where 99% is bound to red blood cells. From here it is redistributed to other areas of the body. In adults 95% of lead is stored in bone as compared to 70% in children. For children the remainder is distributed to many other areas of the body including the liver, kidneys, and brain. Lead's half life in these tissue compartments is prolonged.

Pathophysiology

The effects of lead are widespread. Much of lead's toxicity is in its inherent affinity for the biologically active sulfhydryl groups possessed by vital proteins in the body (receptors, enzymes, etc.). Inhibition or interference with these proteins then leads to clinical symptoms. One example is the inhibition of enzymes involved in heme synthesis. This subsequently leads to anemia. Similar disarray can occur in multiple organ systems with lead toxicity. In developing children, these effects are magnified in a neurologic system that is extensively growing and changing.

Clinical Symptoms

Symptoms are dependent on the degree of exposure - which can correlate with blood lead levels -especially if time has passed for blood lead to equilibrate with the other organ compartments. Below are some general examples of clinical symptoms that one

may expect at a given blood lead level. These are generalizations and may not apply in every case.

10-45 mcg/dL - may appear asymptomatic, but child may have subtle cognitive deficits including: impaired cognition, behavior disorders, impaired fine-motor coordination, poor growth, and hearing deficits.

45-70 mcg/dL - neurologic exam may reveal an irritable or lethargic child. Anemia may be present. Gastrointestinal findings include: intermittent vomiting, abdominal pain ("lead colic"), and anorexia.

>70 mcg/dL - encephalopathy (coma, seizures, ataxia, signs of rising intracranial pressure, loss of developmental skills)

Diagnosis

A careful history and physical along with careful consideration lead toxicity in one's differential are the most important components of the diagnosis. A blood lead level is then drawn, usually capillary first as a screen and then venous as confirmation.

Erythrocyte protoporphyrin levels (free and zinc) use to be used in the past when lead levels were not readily available. A microcytic, hypochromic anemia is suggestive of chronic lead exposure. Basophilic stippling may also be seen with chronic lead poisoning. Acutely, lead foreign bodies may be seen on KUB.

Treatment

Removal from the exposure is the first step. This may require the involvement of medical social work and initial inpatient treatment/evaluation.

For lead level above 45, the CDC recommends chelation therapy. Oral chelation therapy with succimer is the preferred choice for children who are not manifesting signs or symptoms of CNS toxicity and whose levels are less than 70. For children who are manifesting CNS signs or symptoms suggestive of encephalopathy, BAL (British anti-lewisite) should be administered first by intramuscular (IM) injection (mixed with Procaine) and then followed 4 hours later by EDTA (edetate calcium disodium). BAL is IM only, is suspended in peanut oil, is eliminated by both renal and biliary excretion, and fully crosses the blood brain barrier (it is the only parenteral chelator that crosses the blood brain barrier).

Exposure to lead may occur in several ways:

- (1) Eating foods or drinking water that contain lead.
- (2) Spending time in areas where leaded paints have been used and are deteriorating.
- (3) Working in jobs where lead is used.
- (4) Using health-care products or folk remedies that contain lead.
- (5) Engaging in hobbies in which lead may be used such as sculpturing (lead solder) and stained glass.

Children are especially vulnerable to lead poisoning. The Virginia Department of Health recommends that children at risk for lead poisoning be screened following the guidelines set forth by the Center for Disease Control and the Virginia Department of Health.

Dr. Christopher Holstege, M.D.
Co-Director, Div. of Medical Toxicology, University of Virginia
Medical Director, Lead Safe Virginia, VA Dept. of Health