CLINICAL VIGNETTE

Lead Toxicity

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A 29-year-old woman presented with history of episodes of syncope for the preceding two years. She had onset of gastrointestinal cramps, headaches, and new onset of seizures one year before. She was admitted to the hospital with abnormal EEG, normal head CT, and normal brain MRI. Labs showed low hemoglobin. She lived in an old house undergoing renovations and had been exposed to paint dust. Environmental testing showed high lead level in the home, and she moved out several months before coming in for evaluation.

Physical examination was normal. Labs revealed an elevated lead level of 30 with normal CBC and comprehensive metabolic panel.

Occupational health was consulted and did not feel chelation therapy was needed. She continued to have lead level monitoring, and they decreased to normal range after 4 months.

Elevated lead level is defined as greater than or equal to 10 mcg/dl based on the CDC's Adult Blood Lead Epidemiology and Surveillance (ABLES) program.[1]

Lead exposure in the form of inorganic lead can occur from soil, water, pottery, household dust, toys, herbal remedies, and traditional cosmetics. It occurs most commonly from occupational exposure, mainly in battery manufacturing, lead and zinc ore mining, and paper and painting industries. Lead content of paint was unregulated until 1977, leading to exposure in the construction industry. Lead from lead-based paint may also be released through past or ongoing home renovation. Lead-contaminated household dust is the major course of lead exposure to children in the U.S.[2] Organic lead was a component in leaded gasoline but was eliminated from gasoline in the U.S. in 1976. It may be a source of lead toxicity in countries that still use leaded gasoline. Organic lead is more toxic than inorganic lead because the body absorbs it more easily.

Lead poisoning has been reported in pregnant women using traditional Ayurvedic medications from India as herbal supplements.[3] Lead can cross the placenta, and lead toxicity is associated with miscarriages, stillbirths, and preterm deliveries. Babies have been noted to have lower birth weight and cognitive impairments.[4,5]

Lead is absorbed through the lungs or the gastrointestinal tract. In adults, the exposure occurs mostly via the respiratory tract. Gastrointestinal absorption in adults is less common than in children; about 20% of ingested lead is absorbed and is usually due to working or eating in a lead contaminated environment. Absorption is increased during fasting. GI absorption is the more common route of exposure in children, about 50%.[6]

After absorption, 99% of the lead in blood is bound to the erythrocyte; 1% remains in the plasma and circulates to the soft tissue. The bones and teeth of adults carry 94% of the total body lead; in children, it is about 73%.[7] Lead accumulation will occur predominately in trabecular bone during childhood and in both cortical and trabecular bone in adulthood.[8] Lead deposits in the skeleton and has a half-life of decades.[9] Bone-to-blood lead mobilization increases during periods of pregnancy, lactation, menopause, physiologic stress, chronic disease, hyperthyroidism, kidney disease, broken bones, and advanced age, all which are exacerbated by calcium deficiency.[10,11]

The nervous system is most at-risk from lead exposure. In children, acute symptoms include ataxia, coma, seizures, and hyperirritability. Persistent effects include IQ changes, ADHD, and hearing impairment.

In adults with chronic exposure and lead level between 40-120, the symptoms can include decreased libido, mood changes, headache, decreased cognitive performance, irritability, and paresthesia. Slowed nerve conduction and wrist drop can occur with chronic high lead levels.[12-14]

Lead in the circulation is excreted by the kidneys. The half-life is estimated to be about 30 days.[9] With acute high-dose lead exposure, renal function may exhibit aminoaciduria, glycosuria, hyperphosphaturia, and Fanconi syndrome. The effect is reversible. With chronic exposure, chronic lead nephropathy (chronic interstitial nephritis) can occur and lead to gout and hypertension.[15]

Acute high lead level can lead to hemolytic anemia and chronic lead exposure can cause hypochromic, normo, or microcytic anemia.[15]

Other effects of lead toxicity include abdominal pain in severe exposure, decreased sperm count, and developmental effects.

Blood lead level is used for evaluation of lead exposure. If anemia is present, a peripheral blood smear can screen for basophilic stippling, which indicates a high level of lead.

Other tests include x-ray fluorescence, which measures the bone lead concentration and reflects cumulative lead exposure. Currently, x-ray fluorescence is limited in availability.

Management of lead toxicity will depend on the blood lead level.

Due to concern of long-term lead effects, the goal is to keep blood lead level less than 20 mcg/dL and ideally below 10 mcg/dL. For pregnant women, the goal is less than 5 mcg/dL.[16,17]

For the level between 10-19 mcg/dL, management includes lead education and referrals to occupational medicine and appropriate agencies. Testing should be repeated every 2-3 months. If the repeat level persists in the 10-19 range, aggressive environmental intervention will be performed.

For the range between 20-44, patients should continue follow-up testing and have a clinical evaluation for management of any symptoms and start aggressive environmental intervention.

For the range between 45-69, treatment includes education, clinical evaluation, and management within 48 hours, as well as providing appropriate chelation therapy. For level > 70, or in case of encephalopathy, patient will be admitted for immediate chelation therapy.[15]

Chelation agents can decrease the blood level of lead rapidly to relieve symptoms. Chelation is started for levels greater than 100 mcg/DL, can be started in patients with lead symptoms and level 50-80 mcg/dL, and in certain cases of asymptomatic patients with level 80-100 mcg/dL. The most common agents include DMSA and Ca EDTA.

Calcium EDTA is administered IV or IM. Patients with normal renal function are given CaEDTA in a hospital setting with close monitoring for renal function due to renal toxicity. DMSA (2,3-dimercaptosuccinic acid, succimer) is an oral chelation agent that is approved for treatment of children with lead poisoning and can be used in adults. The dose is 10 mg/kg, 3 times/day for 5 days, then 10 mg/kg twice/day for 2 weeks. Side effects include nausea, rash, elevated LFTs, neutropenia, and abdominal discomfort.[18,19]

Patients will need to take ongoing steps to minimize the exposure at home and in the workplace, as well as continue to have follow-up lead testing for continued monitoring.

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