


دوتنه تخصصی پښتني فانونی او مسسور پښت ها  
دکتور مریم رحیمی  
مسسور پښت جا سر بر

## **PHARMACOLOGY**

**Absorption of inorganic lead is usually by the respiratory and GI tracts; skin absorption is negligible. Dietary deficiencies in calcium, iron, copper, and zinc may contribute to increased GI absorption in children.**

**Greater than 90% of the total body lead is stored in bone, where it easily exchanges with the blood. Lead can be transferred across the placenta, a process exacerbated by increased bone turnover during pregnancy. Excretion of lead occurs slowly; the biologic half-life of lead in bone has been estimated to be 30 years.**



## **PATHOPHYSIOLOGY**


**Lead toxicity primarily affects the nervous, cardiovascular, hematopoietic, and renal systems**




## **CLINICAL FEATURES**

**Young children are more susceptible than adults to the effects of lead.**

**Encephalopathy, a major cause of morbidity and mortality, may begin dramatically with seizures and coma or develop indolently over weeks to months with decreased alertness and memory progressing to mania and delirium.**



**Encephalopathy due to lead poisoning typically occurs in toddlers ages 15 to 30 months old with blood lead levels >100 micrograms/dL but has been reported with blood lead levels of 70 micrograms/dL or lower.**



seen as "lead lines" on radiographs of long bones. Lead-induced adverse effects on the reproductive system include increased fetal weight, rupture of membranes, depressed sperm counts, abnormal or nonmotile sperm, and sterility.

## Clinical Features


The common signs and symptoms of lead toxicity primarily vary according to the type of exposure (acute vs. chronic) and, to a lesser extent, to the age of the individual and type of lead (inorganic vs. organic) involved (**Table 197-2**). Young children are more susceptible than adults to the effects of lead. Encephalopathy, a major cause of morbidity and mortality, may begin dramatically with seizures and coma or develop more gradually over weeks to months with decreased alertness and memory progressing to mania and delirium.<sup>11</sup> Encephalopathy due to lead poisoning typically occurs in toddlers ages 15 to 30 months old with blood lead levels >100 micrograms/dL but has been reported with blood lead levels of 70 micrograms/dL.

**Table 197-2 Common Manifestations of Lead Poisoning**

System	Clinical Manifestations
Central nervous system	Acute toxicity: encephalopathy, seizures, altered mental status, papilledema, optic neuritis, ataxia
	Chronic toxicity: headache, irritability, depression, fatigue, mood and behavioral changes, memory deficit, sleep disturbance
Peripheral nervous system	Paresthesias, motor weakness (classic is wrist drop), depressed or absent deep tendon reflexes, sensory function intact

# **DIAGNOSIS**

**History of an exposure, whether occupational or environmental or related to a hobby or retained lead bullet, is the most important clue in making the diagnosis. The clinician should focus on symptoms, developmental and dietary histories (in children), pica, any house or day care remodeling, previous serum iron and whole-blood lead levels, and possible lead toxicity in other family members.**





**The combination of abdominal or neurologic dysfunction with a hemolytic anemia should raise the suspicion of lead toxicity. Consider the diagnosis in all children presenting with encephalopathy.**




**The blood lead level is the best single test for evaluating lead toxicity, and levels at or >10 micrograms/dL are considered elevate**



## **DIFFERENTIAL DIAGNOSIS**

The differential diagnosis of lead toxicity includes causes of encephalopathy, such as Wernicke encephalopathy, withdrawal from ethanol and other sedative-hypnotic drugs, meningitis, encephalitis, human immunodeficiency virus infection, intracerebral hemorrhage, hypoglycemia, severe fluid and electrolyte imbalances, hypoxia, arsenic, thallium, and mercury toxicity, and poisoning with cyclic antidepressants, anticholinergic drugs, ethylene glycol, or carbon monoxide.

**The abdominal pains of lead toxicity can mimic sickle cell crisis or the hepatic porphyrias. Chronic lead toxicity can mimic major depression, hypothyroidism, polyneuritis, gout, iron deficiency anemia, and learning disability.**



# TREATMENT

Lead-induced encephalopathy is rare, but it remains capable of serious morbidity and mortality. In severely toxic patients, standard life-support measures should be instituted. Seizures are treated with benzodiazepines, phenobarbital, and general anesthesia, if necessary.

**Lumbar puncture may precipitate cerebral herniation and should be performed carefully, if at all, with the removal of only a small amount of cerebrospinal fluid. If lead encephalopathy is suspected, chelation therapy should be promptly instituted (i.e., in the ED) without waiting for the results of a blood lead level.**



Chelation therapy for lead toxicity uses dimercaprol (previously known as *British anti-Lewisite* or *BAL* ), edetate calcium disodium (sometimes abbreviated  $\text{CaNa}_2\text{EDTA}$ ), and succimer (also known as *dimercaptosuccinic acid* or *DMSA* ).