Lead poisoning in children can be defined as a blood lead level of 10 µg/dL or more or 5 µg/dL or more in a child with suspected or documented exposure to lead. The most common sources of lead include lead-based paint, batteries, and leaded-gasoline. Symptoms of lead poisoning include constipation, abdominal pain, vomiting, loss of appetite, and impaired neurobehavioral performance. Learning difficulties are common. Lead encephalopathy presents with symptoms and signs suggestive of increased intracranial pressure and can be complicated by cerebral edema. Chelation therapy is indicated in children with blood lead levels of 45 µg/dL or more.

Overview

Lead poisoning in children is a very common problem in the United States and all over the world. Elevated blood lead levels are commonly seen in children and have been linked with mental retardation and growth failure.

Elevated blood lead levels can come from exposure to old buildings that were painted
with lead-containing paint in the pre-1979 period. Other possible sources of lead poisoning in children include exposure to batteries, cement, cosmetics, and chewing on imported painted toys that have lead in the paint. Lead poisoning is defined as a blood lead level that is equal to or more than 10 µg/dL.

Epidemiology of Lead Poisoning in Children

The estimated prevalence of elevated blood lead level of 10 µg/dL or more in children younger than 72 months in the United States is around 0.56%. When a less-strict definition of lead toxicity is used with a lower cut-off of 5 µg/dL or more, approximately 4 million children in the United States are found to have lead toxicity.

Children who live in old houses with deteriorating paint that were built before 1979 are at an increased risk of having an elevated blood lead level.

In other areas of the world where leaded gasoline and lead-based paint is still used, the incidence and prevalence of lead poisoning in children is significantly higher.

Lead poisoning is rarely observed in children who are older than 6 years of age. Elevated blood lead levels are more commonly seen in African Americans and Hispanic children compared to white children.

The prognosis of children who are confirmed to have elevated blood lead levels is usually dependent on the severity of lead toxicity in the child and on the severity of the symptoms at presentation.

Asymptomatic children with lower blood lead levels usually have a good prognosis with improved intellectual function after further lowering the blood lead levels. Unfortunately, children with markedly elevated blood lead levels and severe neurologic damage do not show any intellectual improvement even after the initiation of treatment.

Pathophysiology of Lead Poisoning

Children can be exposed to organic or inorganic lead, but most cases of lead toxicity are due to exposure to inorganic lead. Elevated blood lead levels can occur after ingestion of lead, inhalation exposure to sources of inorganic lead or transdermal absorption after prolonged exposure to possible sources of inorganic lead such as lead-based paint.

Our body’s ability to absorb lead depends on the size of the particles, the presence of other nutritional deficiencies in the child, and certain dietary habits. Exposure to fine dust from lead-based products or to leaded gasoline puts the child at a significantly higher risk of absorbing lead and developing lead toxicity compared to exposure to larger particles in size.

Children with iron, calcium, zinc, and protein deficiencies usually have a higher absorption of lead compared to healthy children. Finally, children who have excessive fat intake have increased lead absorption, while children who eat a healthy diet that is rich in leafy green vegetables have improved lead elimination.

Once the lead is absorbed, it inhibits sulphydryl-dependent enzymes. Lead is usually deposited in the bone and soft tissues. Lead toxicity is associated with impaired hematopoiesis, decreased heme synthesis, and the accumulation of cellular toxic
compounds such as aminolevulinic acid.

Lead can also induce neural cell damage by the accumulation of certain toxic compounds within the central nervous system. This usually manifests as impaired cognitive function and decreased academic performance in children. In severe cases of lead toxicity, profound mental retardation can be seen.

Clinical Presentation of Lead Poisoning in Children

![Image: “Dense metaphyseal lines from lead poisoning.” by Dr Abhijit Datir. License: CC BY-SA 3.0](image)

The symptoms of lead poisoning in children are non-specific. Any child who presents with anorexia, vomiting, constipation and abdominal pain without any identifiable cause and with possible exposure to lead should be evaluated for the possibility of lead poisoning.

Children with lead poisoning can develop symptoms and signs of central nervous system involvement or peripheral nervous system disease. Peripheral neuropathy due to lead poisoning in children is rare. The most common features of central nervous system involvement include attention deficits and learning problems. Children can develop symptoms of peripheral neuropathy, such as peripheral nerve palsies, but this is rarely seen.

Children with lead poisoning usually have impaired hematopoiesis which causes anemia; therefore, pallor, breathlessness, and tachycardia can be seen early in the disease. Children with severe lead poisoning can develop increased intracranial pressure, which can present with a decreased level of consciousness, bradycardia, hypertension and respiratory depression.

Children with symptoms and signs suggestive of severe central nervous system involvement and elevated intracranial pressure should undergo a funduscopic examination of the eye to confirm the presence of papilledema.
Diagnostic Workup for Lead Poisoning in Children

Nowadays, routine and universal screening for lead toxicity in children aged between 1 and 2 years is no longer recommended. Instead, social service providers should identify children with increased environmental risk of exposure to lead and the assessment of blood lead levels should be performed in this cohort only.

Determination of the plasma glucose level and serum electrolytes in any child with an impaired level of consciousness is essential. Additionally, the determination of calcium levels in the blood is useful because of the increased absorption of lead in children with calcium and other nutritional deficiencies.

The diagnosis of lead toxicity in children can be confirmed only by measuring the blood lead level. The blood lead level is essential for guiding the treatment approach for the child. Classically, lead poisoning is confirmed when the levels of blood lead exceed 10 µg/dL.

More recently, the Centers for Disease Control and Prevention (CDC) have suggested using a lower cut-off of 5 µg/dL to confirm the diagnosis of lead poisoning in children. A blood lead level of more than 45 µg/dL is indicative of severe lead poisoning and is an indication for chelation therapy.

A complete blood count should be obtained in children with confirmed or suspected lead poisoning. The most common finding on a complete blood count is hypochromic microcytic anemia. Erythrocyte basophilic stippling is rarely seen in children with lead toxicity compared to adults.

Children with a blood lead level of 55 µg/dL or higher should undergo erythrocyte protoporphyrin testing to determine the levels of this toxic compound and the degree of impairment in hematopoiesis.

Children with chronic exposure to lead can have increased lines of radiodensity at the distal metaphyseal zone of their long bones on an X-ray. Children with an altered level of consciousness or focal neurological signs should undergo a head computed tomography scan or a magnetic resonance imaging study to exclude the presence of cerebral edema.

### Evidence of Lead Poisoning

<table>
<thead>
<tr>
<th>5-14</th>
<th>15-44</th>
<th>45-69</th>
<th>&gt; 70</th>
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</thead>
<tbody>
<tr>
<td>Remove exposures</td>
<td>Confirm venous sample within 1 month</td>
<td>Usually asymptomatic</td>
<td>Encephalopathy</td>
</tr>
<tr>
<td>Retest in 3 months</td>
<td>AXR if pica</td>
<td>Chelation with oral succimer</td>
<td>Hospitalize</td>
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<tr>
<td>Associated with learning deficits</td>
<td></td>
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<td>Succimer and CaNa₂EDTA</td>
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### Treatment of Lead Poisoning in Children

Treatment of lead poisoning in children is usually restricted for blood lead levels that are above 45 µg/dL. Children with lower levels and without radiographic findings suggestive of chronic lead exposure might benefit from prevention of future lead exposure and decontamination alone without chelation therapy.
Children with blood lead levels between 45 and 69 µg/dL can be managed in an outpatient setting as long as further exposure to lead is guaranteed to be eliminated. When in doubt, the child should be admitted for inpatient treatment.

Children with confirmed lead toxicity who have blood lead levels of 70 µg/dL or more should receive the chelator dimercaprol followed by calcium disodium edetate. Use of calcium disodium edetate alone increases the risk of lead re-distribution to the central nervous system due to its limited ability to cross the blood-brain barrier.

Children with acute exposure to lead, such as the ingestion of batteries, can undergo gastric lavage or whole-bowel irrigation. Unfortunately, the efficacy of these therapeutic options is not confirmed.

Children who are confirmed to have iron deficiency in addition to lead poisoning should not receive iron replacement therapy while on dimercaprol because of the increased risk of toxicity.

Children with severe neurological symptoms, i.e. lead encephalopathy, might develop seizures. Benzodiazepines should be used to treat seizures in these children. Due to the altered level of consciousness and depressed respiratory drive, children with lead encephalopathy should undergo endotracheal intubation.

Hyperventilation or mannitol can be used to decrease the intracranial pressure in children with lead encephalopathy. Corticosteroids might be beneficial in children with confirmed cerebral edema.

Aspirin Toxicity

Pathology

- Fever and metabolic acidosis due to uncoupled oxphos
- Central induced tachypnea, respiratory alkalosis
- Increased HR
- Agitation to coma in severe cases
- Pale and diaphoretic
- Vomiting, acidotic, hypoglycemia

Aspirin involved in:

- “Pepto-bismol”
- Wart remover medication
- Herbal remedies

Management

ABC’s

- Care with intubation and acid/base status management
- Correct hypokalemia and hypoglycemia
- GI decontamination
- Alkalinize urine to improve ASA excretion
References


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