



## A REVIEW: LEAD TOXICITY AND IT'S MEDICOLEGAL ASPECTS

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### ABSTRACT

Lead is a metallic inorganic irritant poison. According to Health and Human Services Department USA, lead poisoning is the most important environmental problem. Poisoning is more common from chronic occupational exposure among lead smelters, battery manufacturers, painters, decorators, etc. Lead is a cumulative poison, remains accumulated in bones as phosphate and carbonate. Minute quantities of lead are always present in the blood of even normal individuals. Only when the concentration is high, do features of intoxication begin to manifest. Today the accepted upper level for blood lead (BL) is fixed as 35 mcg/100 ml. Chronic exposure may also occur at home from paint, pottery and contaminated drinking water by lead pipes used for city water supply.

**KEYWORDS:** lead toxicity, poisoning, acute, chronic, exposure etc.

### INTRODUCTION

Lead is a soft, blue-gray metal that is mined from the earth's crust. Human exposure to lead and its compounds occurs mostly in lead related occupations with various sources like leaded gasoline, industrial processes such as smelting of lead and its combustion, pottery, boat building, lead based painting, lead containing pipes, battery recycling, grids, arm industry, pigments, printing of books, etc.<sup>[1]</sup> Lead is the most important toxic heavy element in the environment. Due to its important physico-chemical properties, its use can be retraced to historical times. Metallic lead has been part of the human environment for over 5000 years, and is today detectable in practically all phases of the inert environment and in all biological systems worldwide. Because of its malleability and low melting point, it was one of the first metals smelted and used by early human societies. Hippocrates is credited with the earliest description of chronic lead poisoning when he associated persistent abdominal colic in a man, with his occupation of

extracting metals (around 370 BC). The ancient Romans were however the first to experience the metal's adverse effects on a massive scale mainly because of chronic poisoning through lead acetate which was used to sweeten wine in those days. Chemical analyses of the bones of Roman rulers have demonstrated high lead content and the madness of some of the Roman aristocracy (*Nero, Caligula*), may actually have been the result of lead poisoning.<sup>[2]</sup> Lead is an industrial poison, it's poisoning is mainly accidental and usually chronic.

### Physical Appearances<sup>[3]</sup>

Elemental lead exists as a highly lustrous, heavy, silvery-grey metal with a cubic crystal structure that assumes a bluish tint as it tarnishes in air. It is quite soft and malleable. Several of its salts occur as variously coloured powders or liquids and are used widely in industry and at home producing cumulative toxicity on chronic exposure.

### Toxic Compounds and Its Uses<sup>[4]</sup>

Compounds	Uses
Lead acetate (sugar of lead)	Earlier used as an astringent and local sedative for sprains
Lead tetraoxide (red lead or <i>vermilion</i> )	Used as <i>sindoor</i>
Tetraethyl lead	Antiknock for petrol
Lead sulfide ( <i>surma</i> ; least toxic)	Applied on the eyes
Lead carbonate (white lead)	Manufacture of paints

**Mode of Action**<sup>[5]</sup>

1. Lead combines with sulfhydryl enzymes leading to interference with their action.
2. It decreases haeme synthesis by inactivating the enzymes involved such as *aminolaevulinic acid dehydrase*, *aminolaevulinic acid synthetase*, *coproporphyrinogen oxidase* (or *decarboxylase*), and *ferrochelatase*. This results in anaemia.
3. Lead increases haemolysis as a result of which immature red cells are released into circulation such as reticulocytes and **basophilic stippled cells** (the result of aggregation of ribonucleic acid due to inhibition of the enzyme pyrimidine-5-nucleotidase which normally eliminates degraded RNA)
4. In the CNS, lead causes oedema and has a direct cytotoxic effect leading to decreased nerve conduction, increased psychomotor activity, lower IQ, and behavioral / learning disorders. Children are especially susceptible. The highest brain concentrations of lead are found in hippocampus, cerebellum, cerebral cortex, and medulla.
5. Lead also has deleterious effects on the CVS (hypertension and myocarditis), kidney (nephritis), and reproductive organs (infertility). Lead nephropathy after chronic lead exposure has been well described. Interstitial nephritis, reduced glomerular filtration rate, and nonspecific proximal tubular dysfunction are typical. In addition, lead can decrease uric acid renal excretion, thereby raising blood urate levels and predisposing to gout (*saturnine gout*). Elevated urinary levels of N-acetyl-3-D-glucosaminidase and beta-2-microglobulin may serve as early markers of renal injury.
6. **Permissible lead intake and blood levels**
  - a. There is much controversy over this at the present time. Lead appears as a trace metal in virtually all foods and beverages, though fortunately absorption from such sources is relatively low.
  - b. Adults ingest 300 mcg and inhale 15 mcg of lead approximately each day, of which only 10% is absorbed, but children may absorb upto 50%.
  - c. An important source of food based lead poisoning is the use of lead-soldered canned food and drink. While in USA measures have been taken to ban lead soldering of cans, the Indian canned food industry may still be persisting with lead soldered seams, though there is no clear information on this.
  - d. Lead in drinking water may be absorbed to greater extent than that in food. The concentration of lead which may not really be very high in ground or surface water may progressively rise as it passes through the distribution system because of contact with lead connectors, lead service lines or pipes, lead soldered joints, lead containing coolers, and lead impregnated fixtures such as brass taps.
  - e. Because of the reasons mentioned, minute quantities of lead are always present in the blood of even normal individuals. Only when the concentration is

high, do features of intoxication begin to manifest. Today the accepted upper level for blood lead (BL) is fixed as 35 mcg/100 ml. However there are reports that adverse effects especially on the haematopoietic system can occur at levels as low as 10 mcg/100 ml. Neurobehavioral disorders in children can occur at BL as low as 25 mcg/100 ml. Hence, the current trend is to consider even levels as low as 10 mcg/100 ml as unacceptable, especially in children.

**Absorption and Excretion**<sup>[6]</sup>

- Lead is absorbed through the GIT, respiratory tract (dust and fumes) and skin (lead tetraoxide). In blood, 95–99% of lead is sequestered in RBCs.
- Absorption of lead compounds is directly proportional to solubility and inversely proportional to particle size. GIT lead absorption is increased by iron deficiency and low dietary calcium, and decreased by ingestion with food.
- It is a cumulative poison. In chronic exposure, it deposits in tissues, mostly in the bones (90%), liver and kidneys.
- It is mainly excreted through the urine (70%), but rate of excretion is low; smaller amounts are eliminated via feces, and scant amounts via the hair, nails and sweat.

**Signs and Symptoms**

Lead poisoning clinically manifests in two forms: acute and chronic forms.

**Acute Poisoning**<sup>[7]</sup>

It manifests as GIT and CNS disturbances.

- **GIT:** Metallic taste, dry throat, thirst, vomiting, nausea, burning abdominal pain (colic) and blood stained diarrhea leading to circulatory collapse.
- **CNS:** Headache, lethargy, arthralgia, myalgia, anorexia, insomnia, paresthesia, depression, coma and death.

**Sub-acute Poisoning**<sup>[8]</sup>

- This type of poisoning occurs from repeated small doses of lead acetate.
- Blue line on the gums is seen with gastrointestinal symptoms.
- Urine is scanty and in deep red colour.
- In the later stages, nervous symptoms become prominent with numbness, cramps and flaccid paralysis of lower limbs.

- **Fatal dose** depends on toxic compound  
20 gm of lead acetate  
40 gm of lead carbonate.

- **Fatal period** 1 to 2 days.

**Laboratory diagnosis**<sup>[9]</sup>

1. Porphyrinuria due to coproporphyrin III.
2. Blood lead level > 70–100 µg/dl. Protoporphyrin > 35 µg/dl.
3. Urine lead level > 0.15–0.3 mg/l.

**Treatment of Acute and Subacute Poisoning**<sup>[10,11]</sup>

- Emetics
- Stomach wash with 1 % magnesium or sodium sulphate solution followed of ample washing with plain water to remove the lead sulphate formed.
- 25 gm of magnesium sulphate orally with demulcent drinks
- Calcium gluconate 1 gm to relieve colic
- Intravenous fluids
- Chelating agents like EDTA, BAL and penicillamine are helpful.
- A diet rich in milk, administration of calcium salts and vitamin D are helpful.
- Peritoneal or haemodialysis may be necessary.

**Chronic Poisoning (Plumbism, Saturnism)**<sup>[12,13,14]</sup>

It was also called *colica pictorum*, *painter's colic*. Chronic poisoning with lead compounds manifests with a set of symptoms, which may be enumerated as:

**1. Facial pallor:** Pallor seen especially around the mouth also known as *circum oral pallor* is due to the *vasospasm* of the capillaries and arterioles, around the mouth.

**2. Anemia:** In early stages, there may be polycythemia with polychromatophilia, but later there is anemia with karyorrhesis and dyserythropoiesis (*punctuate basophilia, reticulocytosis, poikilocytosis, anisocytosis*), nucleated red cells and increase in mononuclear cells in peripheral blood and ringed sideroblasts in bone marrow.<sup>[4]</sup> However, polymorphonuclear cells and platelets are decreased. RBC count comes down to 3.5 million/dl and hemoglobin level to 6.5 g%.

**Cause of anemia**

- Impairment in heme synthesis from protoporphyrin and of porphobilinogen from δ amino levulinic acid.
- Increased fragility of RBCs due to loss of intracellular potassium (there is an increased permeability of cell membrane to K<sup>+</sup>).

**3. Burtonian line (lead line):** It is a stippled blue line seen at the junction of the gums usually nearer to a tooth caries, especially in the upper jaw. This is due to the deposition of lead sulphide formed by the action of the combination of lead with hydrogen sulphide which had evolved from the decomposed food debris in the caries tooth.

**4. Lead colic and constipation:** The victim will complain of severe colicky pain abdomen relieved by pressure and bowel irregularities. Abdominal muscles become tense and retracted.

**5. Lead palsy:** There is a typical paralysis affecting the extensor muscles of the fingers and wrist causing '*wrist drop*' and '*claw shaped hand*'. Similarly paralysis may extend to the extensor muscles of the foot leading to foot drop.

**6. Lead encephalopathy:** Mostly seen in infants presenting with severe ataxia, vomiting, lethargy, stupor, convulsion and coma. Cerebral psychic affection may be present.

**7. Cardiorenal manifestations:** Elevated blood pressure and arteriosclerotic changes are observed. Urine contains albumin and abnormal quantity of lead, coproporphyrin III and delta amino laevulinic acid. Interstitial nephritis may occur.

**8. Reproductive system:** menstrual derangements, such as amenorrhoea, dysmenorrhoea, menorrhagia, sterility of both sex and abortion are frequent. Abortion occurs in pregnant women between 3-6 month.

**Laboratory diagnosis of chronic lead poisoning**<sup>[15]</sup>

- Urine lead levels of more than 0.08 mg per litre collected in 24 hours
- Blood lead level more than 0.8 mg per litre
- Increased urine and plasma delta-amino laevulinic acid, Increased coproporphyrin level in urine
- X-ray evidence of increased density or radio opaque bands or lines at the metaphyseal ends of long bones in children. This is also referred to as *lead lines*.
- Presence of lead as radio opaque material on X-ray stomach and intestines may be seen in children particularly with history of pica (meaning abnormal craving for non-nutritive substances).

**Treatment**<sup>[16]</sup>

1. Remove the patient from the source of exposure.
2. Potassium or sodium iodide 1–2 g TDS orally.
3. Sodium bicarbonate 20–30 g in 4 or 5 divided doses orally.
4. MgSO<sub>4</sub> or sodium sulfate 8–12 g orally.
5. **CaNa<sub>2</sub>EDTA** IV in usual doses. Chelation therapy is indicated for adults with blood lead > 70 µg/dl and for children with encephalopathy or blood lead > 45 µg/dl.
6. **BAL:** Chelator of choice in case of renal impairment. Succimer (DMSA) is given in mild to moderate toxicity in a dose of 10 mg/kg orally every 8 h for 5 days, then every 12 h for 2 weeks.
7. Correction of dietary deficiencies in iron, calcium, magnesium and zinc lowers lead absorption. Vitamin C may be added. Iron supplementation is withheld during chelation therapy.
8. Ammonium chloride 1 g, 3–4 times given daily. By this, lead deposited in the bones is mobilized into the blood and excreted.
9. Mannitol for cerebral edema, and diazepam IV for seizures associated with lead encephalopathy; hemodialysis in cases of renal failure.
10. Symptomatic treatment.

**Postmortem Changes**<sup>[17,18]</sup>**Acute Poisoning**

**External:** Nothing specific.

**Internal:** Stomach-gastric mucosa is congested, eroded and patchy in appearance with greyish white deposits. Large intestine may show black colored faecal matter. Evidence of renal tubular degeneration.

**Chronic Poisoning**

Blue line on the gums. Muscles are flaccid and show fatty degeneration. Intestines are contracted and thickened. Liver and kidneys are hard and contracted. Heart is hypertrophied. Renal tubular necrosis is usually noticed. Brain is very pale and swollen. Bone marrow shows hyperplasia of leucoblasts and erythroblasts with a decrease in fat cells.

**Medicolegal Importance**<sup>[17,19]</sup>

1. Lead is an industrial poison presenting as an occupational hazard. It is commonly used in industries concerning manufacturing of battery cell, paints, crayon, hair dyes, toys, etc. poisoning is mainly accidental and usually chronic. Accidental poisoning by contamination of the drinking water occurs in places where lead pipes are used. Drinking fruit juices or water stored in improperly glazed ceramic wares can result in lead poisoning.
2. Use of Lead for suicide or homicide is very rare.
3. In cases of long standing gun-shot bullet's lodged and retained in the body, have reported of chronic lead poisoning due to absorption of lead particles from these bullets. Gunshot wounds in adults and children have been reported of causing anorexia, abdominal pain, vomiting, anemia, encephalopathy, seizures, etc. The surface area of retained lead particles, location of retained lead particles (especially synovial fluid), length of time for which one is exposed to lead and type of activation (uncoated bullets-yielding greater surface area of lead for dissolution) are all factors that may lead to lead poisoning.
4. Long standing use of cosmetics containing lead salts (*surma, sindoor, vermilion*, etc) can result in chronic lead poisoning.
5. Sometimes used as cattle poison and to procure abortion.

**DISCUSSION AND CONCLUSION**

Lead is absorbed through all portals of entry. Occupational exposure results mainly from inhalation, while in most other situations the mode of intake is ingestion. Tetraethyl lead can be absorbed rapidly through intact skin. Following absorption, it is stored in the bones as phosphate and carbonate. In children about 70% of total body lead is skeletal, while in adults over 95% is in osseous tissues. Lead is drawn to those areas of the skeleton which are growing most rapidly. These include the radius, tibia, and femur, which are the most metabolically active. The hypermineralisation is reflected in the form of densities which are the classic

“lead lines” observed on x-ray. Lead toxicity associated with IQ deficits, behaviour disorders, slowed growth, and impaired hearing. Studies in population blood-lead concentrations have shown a fall by up to 80 per cent in the last twenty years, cases of lead poisoning continue to occur. Pure metallic forms are nontoxic as a steel-grey metal. However, lead compounds can act by producing spasms of the capillaries and arterioles or by fixation of the poison in the tissues such as brain, bones, etc. It can also combine with *sulphydryl enzymes* and interfere with its action. Lead can decrease synthesis of heme leading to anaemia. Lead can destroy nerve cells, myelin sheaths in CNS and also produce cerebral edema. It also exerts toxic effects on kidneys (nephritis) and reproductive system (infertility). Acute and homicidal poisoning is rare. Accidental chronic lead poisoning occurs in people working with lead.

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