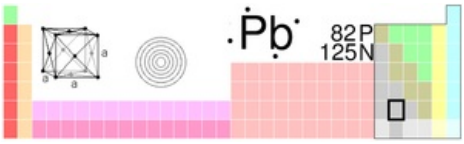
Intoxication by Lead and its compounds

In practice, intoxication with an inorganic or organic form of lead is possible. Inorganic intoxication is much more common, most often in the work environment.



The position of lead in the periodic table

Intoxication by an inorganic form of lead

Lead is a dull gray, heavy, malleable metal (Saturn metal), it dissolves well in gastric juice, its inorganic salts are relatively poorly soluble in water, but the soluble (and therefore more toxic) include oxides, acetate and nitrate.

Car battery

Professional exposure

- Production and repair of **car batteries**,
- **smelting in smelters** (production of lead, bronze, brass),
- when **soldering** (lead-tin alloy),
- production of **lead glass**, cartridges, glazes (PbO) and pigments (Lead(II,IV) oxide, Pb_3O_4),
- rarely from lead glazes on ceramic dishes (hot tea with lemon - releases lead), or swallowing devils (ammunition).

Etiopathogenesis

Lead is absorbed either by respiration or from the GIT;

- **by inhalation** in the form of **vapor and dust** (about **40 %** is absorbed),
- **about 8 %** is absorbed **from the GIT**, it is better absorbed if it stays longer in the stomach, in children it is absorbed by up to 50%.

Higher absorption of lead occurs with calcium and iron deficiency and during starvation. Pb^{2+} ions have two main properties, affinity to SH groups and substitution of Ca^{2+} ions. After absorption, lead is bound to hemoglobin, distributed throughout the body and **deposited in bones, brain** (especially in children), **kidneys, liver**, muscles, skin (and adnexa). The largest proportion of lead is deposited in the bones, where it replaces calcium (in practice, this causes a higher bone contrast on X-ray), from where the lead can be released back into the circulation during fever or pH changes. **80% of lead is excreted with the urine**, the half-life of lead in the blood is 30 days. It is excreted from the bones for 5-10 years.

Free lead in the body inhibits enzymes involved in heme synthesis:

- **5-ALA dehydrogenase** – ALA accumulates,
- **coproporphyrin oxidase** – coproporphyrin accumulates,
- **hemsynthetase** – protoporphyrin and iron accumulate, the result is **normocytic normochromic anemia**, an increase in the level of iron in the serum.

Clinical picture

Symptoms **of acute** lead poisoning:

- after ingestion, symptoms of GIT irritation dominate – vomiting, diarrhea,
- number of diseases.

Symptoms **of chronic** lead intoxication:

- gradual development of anemia – fatigue, exertional dyspnea, apathy, muscle and joint pain,
- gray border on the gums, constipation, saturnine colic (diffuse colic pain in the abdomen) that does not respond well to antispasmodics,
- in more severe intoxications, liver enzymes and bilirubin may be increased (Pb is deposited in the liver),
- **nephropathy** with damage to the proximal tubules occurs rarely (Fanconi syndrome with aminoaciduria, glycosuria and phosphaturia),
- very rarely – saturnine gout, hypertension, neuropathy (most often *the radial nerve* is damaged).

Investigative methods

- **Laboratory examination of blood** – lead level in the blood – **plumbemia** – correlates with the clinical picture of poisoning, permissible limit for workers – 0.4 mg/l, levels up to 0.1 mg/l are common in the population,
- **blood count** – anemia (normochromic, normocytic), basophilic staining in erythrocytes is typical, erythrocytes are fragile,

- **laboratory examination of urine:**
  - 5-ALA and coproporphyrin III in urine - signs of recent lead exposure,
  - monitoring of lead in urine - serves to determine the depot (excretion of more than 2 mg/day means a large amount of Pb in the body).

Thanks to laboratory tests, differential diagnosis is relatively easy, the most important thing is to think about the possibility of lead intoxication. Otherwise, lead poisoning can be confused with anemia of unclear etiology (in case of chronic) or NPB (in case of acute poisoning).

Therapy

The treatment is carried out **with chelating substances** - they chelate lead ions, the chelates are excreted in the urine:

- EDTA – a classic drug, administered in a slow infusion (it is slightly nephrotoxic, therefore 500 ml of physiological solution or 5 % glucose is administered with it),
- DMSA – dimercaptosuccinic acid, applied in tablets, we prefer it mainly for children.

Professionalism assessment

- **Risk of NzP** – increase in BET above the limits without symptoms,
- **notification of intoxication (i.e. NzP)** – when anemia or other damage is detected,
- sometimes it is necessary to distinguish **intentional consumption** from intent to highlight professional poisoning.

Intoxication with an organic form of lead

This form of intoxication is no longer of such importance today, previously mainly **tetraethylol** (anti-knock additive to gasoline). Tetraethylol is a fat-soluble substance, which is why it is **neurotoxic**.

- Toxic **psychosis** is typical - headaches, sleep disturbances, dizziness, blurred vision, auditory hallucinations, later convulsions and coma. Anemia does not occur here.
- Examination of **plumbemia** , which is only slightly increased, is usually not enough, the diagnosis is confirmed only by examination of urine, which reveals plumburia.
- DMSA is recommended **for therapy**, EDTA is ineffective.

Links

Related articles

- Intoxication by mercury and its compounds
- Intoxication with methemoglobinizing substances

References

- BENEŠ, Jiří. *Studijní materiály* [online]. [cit. 24.02.2010]. <http://jirben.wz.cz>.

Literature

- PELCLOVÁ, Daniela. *Nemoci z povolání a intoxikace.* 2. edition. Praha : Karolinum, 2006. 207 pp. ISBN 80-246-1183-X.