

Heavy metal poisoning

Acute heavy metal intoxication is rare. In clinical practice, we most often encounter **lead, arsenic and inorganic mercury** poisoning.

Heavy metals affect different organ systems. At the cellular level, the toxic effects are the result of the binding of the metal and the enzyme → a stable but inactive complex is formed that inhibits vital cellular processes. In case of acute poisoning, they are best demonstrated in urine and blood, in case of long-term exposure in the hair. The treatment uses substances that form **chelates** with heavy metals, which are usually excreted in the urine.

Laboratory diagnosis of heavy metal poisoning

Lead

- **Venous Blood:**
 - > 250 µg/l – laboratory toxic concentration,
 - > 700 µg/l – clinical symptomatology.

Arsenic

- **urine:** > 100 µg/l – indicator of acute poisoning;
- **venous blood:** > 50 µg/l;
- **hair:** > 1 µg/kg – indicator of chronic poisoning.

Mercury

- **urine:** > 150 µg/l,
- **venous blood:** > 35 µg/l,
- **hair:** 120-300 mg/kg.

Therapy

Antidote = dimercaptopropanol

- We dose 5 mg/kg – 6x during the 1st day, 3x on the 2nd day, then 1–2x, we continue according to the concentration of nox in the blood and urine,
- dimercaptopropanol forms complexes with intracellular and extracellular Pb, As, Hg, Au,
- is ineffective with a large amount of heavy metal,
- primarily excreted in the bile → is the drug of choice for renal function impairment and poisoning with inorganic mercury compounds,
- administration of iron is contraindicated because a toxic complex is formed.

DMSA

- It is administered in asymptomatic child poisonings,
- we administer 10 mg/kg i.v. á 8 hours during 5 days, then at an interval of 12 hours,
- then we continue according to the levels of noxy,
- creates chelates with Pb, As, Hg,
- is the drug of choice for poisoning with organic mercury compounds.

Calcium disodium versenate CaNa_2EDTA

- Forms chelates with extracellular lead,
- is it ineffective in Hg, As, Au poisoning,
- the complex is excreted in urine → cannot be used in case of alteration of renal functions.

Lead poisoning

Pharmacology

Inorganic lead is found in soil, dust, battery cells, pottery glazes and lead paint. The source of organic lead is mainly leaded gasoline. Absorbed lead binds to **erythrocytes** and is distributed to target organs → liver, kidneys, brain, bones. **Excretion is very slow with a half-life of about 20 years!**

Pathogenesis

Lead damages the organism through several mechanisms. Interferes with heme synthesis by inhibiting erythrocyte ATPase synthesis, inhibits cholinergic functions and causes degeneration of Schwann cells → leading to demyelination.

Clinic

- Moderate poisoning is manifested by anorexia, fatigue, irritability, constipation and occasional abdominal pain,
- severe poisoning can result in encephalopathy with brain edema, convulsions and impaired consciousness,
- another target organ is the kidneys, where tubular functions are damaged.

Diagnostics

- The level of erythrocyte protoporphyrin is used as a screening test → at values $> 350 \mu\text{g/l}$, it is necessary to determine the lead concentration in venous blood, clinical symptomatology develops at values $> 700 \mu\text{g/l}$, but anemia can appear already at a value $> 250 \mu\text{g/l}$;
- the finding of "lead lines" on the epiphyseal edges of long bones during an X-ray examination will also contribute to the diagnosis.

Mercury poisoning

Pharmacology and pathogenesis

In clinical practice, poisoning by organic mercury compounds occurs very rarely. Mercury compounds are highly fat soluble and can cause irreversible damage to the CNS after ingestion. Poisoning by inorganic compounds most often occurs when vapors are inhaled or mercury salts are ingested. Mercury is already volatile at room temperature and its volatility increases with rising temperature, odorless vapors are quickly absorbed through the alveolar membrane. It is nephrotoxic after ingestion.

Clinic

Clinical picture after acute mercury vapor poisoning:

- vapor inhalation causes dyspnoea, cough, fever, salivation and metallic taste in the mouth;
- lung damage can result in pneumonia, necrotizing bronchiolitis and pulmonary edema;
- nephrotoxicity manifests as proteinuria and hematuria, may result in oligoanuric renal failure.

Clinical picture after subchronic/chronic mercury vapor poisoning:

- subchronic/chronic vapor poisoning manifests as stomatitis, gingivitis, blue lines on gums and CNS disorders → tremor, anxiety, mental anorexia, insomnia, irritability;
- nephritis and peripheral neuropathy may be present;
- acrodynia manifests itself in children < 6 years of age with subchronic exposure to vapor or in recent years after using calomel-containing dental cleaning powders → itching, soreness and erythema of the extremities, splenomegaly, fever, dehydration, electrolyte imbalance disorders, tubular function disorders, hypertension, tachycardia and signs of CNS damage (insomnia, irritability).

Clinical picture after ingestion of inorganic mercury salts:

- GIT symptoms: nausea, vomitus, diarrhea, irritation/burning of the oropharynx, esophagus or stomach with subsequent hematemesis;
- skin symptoms: urtica, vesicular eruption;
- renal symptoms: acute tubular necrosis.

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A piece of lead



Mercury