



Heavy Metal Poisoning

1st Course

Lecture : 10

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Introduction

- Heavy metal poisoning results from accumulation of toxic metals in the body that interfere with essential biological functions.

Most common metals: **Lead, Mercury, Arsenic, and Cadmium.**

Learning Objectives

- By the end of this lecture, students should be able to:
 1. Identify major toxic metals and their sources.
 2. Understand mechanisms of toxicity and clinical manifestations.
 3. Recognize diagnostic and treatment principles, including chelation therapy.

Definition & Overview

- Heavy metals are elements with high atomic weight and density $>5 \text{ g/cm}^3$.

They bind to cellular proteins, inhibit enzymes, and cause oxidative stress and multi-organ dysfunction.

Common Routes of Exposure

1. **Inhalation:** industrial fumes, smelting, batteries.
2. **Ingestion:** contaminated food or water.
3. **Skin absorption:** occupational exposure, cosmetics.
4. **Parenteral:** drugs, medical instruments.

General Mechanisms of Toxicity

1. Enzyme inhibition (e.g., sulfhydryl binding)
2. Oxidative stress and lipid peroxidation
3. Mitochondrial dysfunction
4. Impaired neurotransmission
5. Accumulation in bone, liver, and kidneys



Major Heavy Metals

1. Lead (Pb)

2. Mercury (Hg)

3. Arsenic (As)

4. Cadmium (Cd)

5. Iron and Copper (in overload states)

Lead Poisoning (Plumbism)

Sources:

batteries, paints, pipes, contaminated dust.

Pathophysiology:

inhibits heme synthesis → anemia; interferes with calcium metabolism → neurotoxicity.

Target organs:

nervous system, kidney, bone marrow.

Clinical Features of Lead Poisoning

1. Fatigue, irritability, abdominal pain (“lead colic”)
2. Peripheral neuropathy (wrist/foot drop)
3. Anemia with basophilic stippling
4. Cognitive impairment in children
5. Nephropathy, hypertension

Diagnosis of Lead Poisoning

1. Blood lead level ($>10 \mu\text{g/dL}$ significant)
2. Elevated zinc protoporphyrin
3. Radiologic “lead lines” in bones
4. CBC: microcytic anemia

Treatment of Lead Poisoning

1. Remove source of exposure

2. Chelation therapy:

- EDTA (CaNa_2EDTA)
- Dimercaprol (BAL)
- DMSA (succimer)

3. Supportive therapy (hydration, nutrition).

Mercury Poisoning

Forms: elemental (Hg^0), inorganic (Hg^{2+}), organic (methylmercury).

Sources: broken thermometers, dental amalgams, fish (tuna, swordfish), industrial vapors.

Clinical Features of Mercury Toxicity

1. Tremors, ataxia, memory loss (“mad hatter syndrome”)
2. Gingivitis, excessive salivation
3. Renal tubular damage
4. Fetal neurotoxicity (crosses placenta).

Diagnosis & Treatment of Mercury Toxicity

- Blood and urine mercury levels.
- **Chelation therapy:** BAL, DMSA, or DMPS.
- Avoid chelation in organic mercury until source removal.
- Supportive: fluids, renal monitoring.

Arsenic Poisoning

Sources:

pesticides, contaminated groundwater,
wood preservatives.

Mechanism:

inhibits oxidative phosphorylation →
cellular energy failure.

Clinical Features of Arsenic Poisoning

- **Acute:**
 - vomiting, rice-water diarrhea, hypotension, arrhythmia.
- **Chronic:**
 - hyperpigmentation, Mees' lines in nails, peripheral neuropathy, skin cancer.

Diagnosis & Management of Arsenic Poisoning

- Urine arsenic concentration (most reliable).
- ECG and electrolyte monitoring.
- **Treatment:** BAL (dimercaprol), hydration, activated charcoal (if oral).

Cadmium Poisoning

Sources:

batteries, alloys, cigarette smoke, fertilizers.

Toxicity:

renal tubular damage, osteomalacia (“Itai-Itai disease”), emphysema.

No effective chelator — mainstay is prevention and supportive care.

Iron Toxicity

- Usually from overdose of iron supplements (children).

Stages:

1. GI irritation (vomiting, bleeding)
2. Metabolic acidosis, shock
3. Hepatic failure

Treatment: IV Deferoxamine chelation.

Copper & Wilson's Disease

Excess copper deposition in liver, brain, cornea.

Features:

hepatic cirrhosis, tremor, psychiatric changes, Kayser–Fleischer rings.

Treatment:

Penicillamine or trientine chelation; zinc to block absorption.

Chelation Therapy Overview

Metal	Antidote / Chelator
Lead	EDTA, BAL, DMSA
Mercury	BAL, DMSA
Arsenic	BAL
Iron	Deferoxamine
Copper	Penicillamine



Laboratory Monitoring

1. Blood and urine metal levels
2. Liver and renal function tests
3. CBC and electrolytes
4. ECG for cardiac involvement

Anesthetic & Critical Care Implications

1. Avoid nephrotoxic and hepatotoxic drugs.
2. Monitor acid-base balance.
3. Manage ventilation in neurotoxic cases.
4. Prepare for possible seizures or arrhythmias.

Summary

- Heavy metal poisoning causes multi-system effects through enzyme inhibition and oxidative damage.
- Early recognition, removal of exposure, and chelation therapy are lifesaving.
- Anesthesia technologists must be aware of drug–metal interactions and critical monitoring needs.