Acute Poisoning

Introduction to Pesticide Poisoning



- Pesticide poisoning is a significant and growing concern in clinical toxicology, especially in developing countries like India, where agriculture is a major occupation.
- Pesticides are chemical substances used to kill or control pests in farming, forestry, public health, and domestic settings.
- While they help improve crop productivity and control disease vectors, their misuse, accidental exposure, or deliberate ingestion (e.g., for suicide) can lead to acute or chronic toxicity in humans.
- The World Health Organization (WHO) classifies pesticides as highly hazardous chemicals due to their toxic potential and widespread availability.
- Pesticide poisoning is not only a medical emergency but also a public health issue with environmental, occupational, and regulatory implications.

Classification of Common Pesticides Involved in Poisoning

1. Organophosphorus Compounds (OPCs)

- Widely used insecticides.
- Extremely toxic, common in both accidental and suicidal cases.

2. Carbamates

- Similar action to organophosphates but usually **shorter-acting**.

3. Organochlorines

- Persistent in environment; less common today due to bans or restrictions (e.g., DDT).

4. Pyrethroids

- Synthetic analogues of natural pyrethrins; generally safer, but still cause toxicity in high exposure.

Routes of Exposure

- Dermal contact during spraying or handling (especially in rural/agricultural workers).
- Inhalation of vapors or aerosolized particles.

- Ingestion, either accidental or intentional.
- Ocular exposure (splashes during mixing or spraying).

Mechanisms of Toxicity

- Each class of pesticide exerts its toxic effects through **different mechanisms**:
  - OPCs and carbamates: Inhibit acetylcholinesterase, causing cholinergic overstimulation.
  - Organochlorines: Disrupt neuronal ion channels, leading to seizures and CNS excitation.
  - Pyrethroids: Prolong sodium channel activation, resulting in neurotoxicity.

Clinical Significance

- Pesticide poisoning can present with multisystem involvement—neurological, respiratory, cardiovascular, gastrointestinal—and requires prompt recognition and aggressive management, including antidotes, supportive care, and sometimes ICU admission.
- It is especially important in India and other low-income countries, where:
  - Storage practices are poor.
  - Label warnings may not be understood due to illiteracy.
  - Protective equipment is often not used by farmers.
  - Healthcare access is delayed, increasing fatality risk.

Various Pesticide poisoning

Organophosphorus Compounds (OPCs)

Mechanism of Action:

- Inhibit **acetylcholinesterase (AChE)** enzyme.
- This leads to accumulation of **acetylcholine** at synapses and neuromuscular junctions.
- Causes **cholinergic crisis** (overstimulation of muscarinic and nicotinic receptors).

Clinical Symptoms:

A. Muscarinic Symptoms (Mnemonic: SLUDGE/BBB):

- **S**alivation
- **L**acrimation
- **U**rination
- **D**iarrhea
- **G**astrointestinal upset

- **E**mesis
- **B**ronchorrhea, **B**ronchospasm, **B**radycardia

B. Nicotinic Symptoms:

- Muscle fasciculations
- Weakness
- Paralysis
- Hypertension
- Tachycardia

C. Central Nervous System (CNS):

- Anxiety
- Confusion
- Convulsions
- Coma

Management:

1. Decontamination:

- Remove contaminated clothing.
- Wash skin thoroughly with soap and water.
- Gastric lavage if within 1 hour and patient is stable.
- Activated charcoal may be given.

2. Supportive Care:

- Maintain airway, breathing, circulation.
- Oxygen therapy.

3. Antidotes:

- **Atropine**: Competitively blocks muscarinic receptors.
  - High doses required, titrated until bronchial secretions dry and heart rate normalizes.
- **Pralidoxime (2-PAM)**: Reactivates AChE before the "aging" process.
  - Especially effective if given early (within hours).

4. Other Measures:

- Benzodiazepines for seizures.
- ICU care if respiratory failure develops.

Carbamate Poisoning

Mechanism of Action:

- Similar to organophosphates: **Inhibits acetylcholinesterase**.
- However, binding is **reversible and short-lived** compared to organophosphates.

Clinical Symptoms:

- Very similar to organophosphate poisoning.
- Cholinergic symptoms predominate.
- Usually milder and shorter in duration.

Management:

- **Decontamination**: Same as organophosphates.
- **Supportive Care**: ABCs, oxygen, fluids.
- **Antidotes:**
    - **Atropine** is effective.
    - **Pralidoxime is not routinely indicated** because enzyme inhibition is reversible and short-acting.
- Monitor for symptom resolution.

Organochlorines

Mechanism of Action:

- Interfere with **GABA-mediated chloride channels** in neurons.
- Leads to **neuronal hyperexcitability**.

Examples:

- DDT, Lindane, Heptachlor, Endosulfan.

Clinical Symptoms:

- **CNS stimulation**:
    - Tremors
    - Convulsions
    - Hyperexcitability
    - Dizziness
    - Headache

- **Chronic exposure** may lead to liver and kidney damage, reproductive toxicity, and carcinogenesis.

Management:

1. **Decontamination:**
   - Skin decontamination with soap and water.
   - Activated charcoal if ingestion is recent.
2. **Supportive Care:**
   - Control seizures with benzodiazepines or barbiturates.
   - Maintain hydration and monitor renal function.
   - **Avoid epinephrine or catecholamines**, as they may enhance cardiac toxicity.
   - No specific antidote.

Pyrethroids

Mechanism of Action:

- Modify the **sodium channels** in nerve cells.
- Prolong sodium influx, leading to **hyperexcitation**.

Examples:

- Permethrin, Cypermethrin, Deltamethrin.

Clinical Symptoms:

1. **Mild poisoning:**
   - Paresthesia (burning or tingling)
   - Skin irritation
2. **Systemic toxicity (rare, usually high doses):**
   - Dizziness
   - Nausea
   - Muscle twitching
   - Seizures
3. **Allergic reactions**
   - possible.

Management:

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