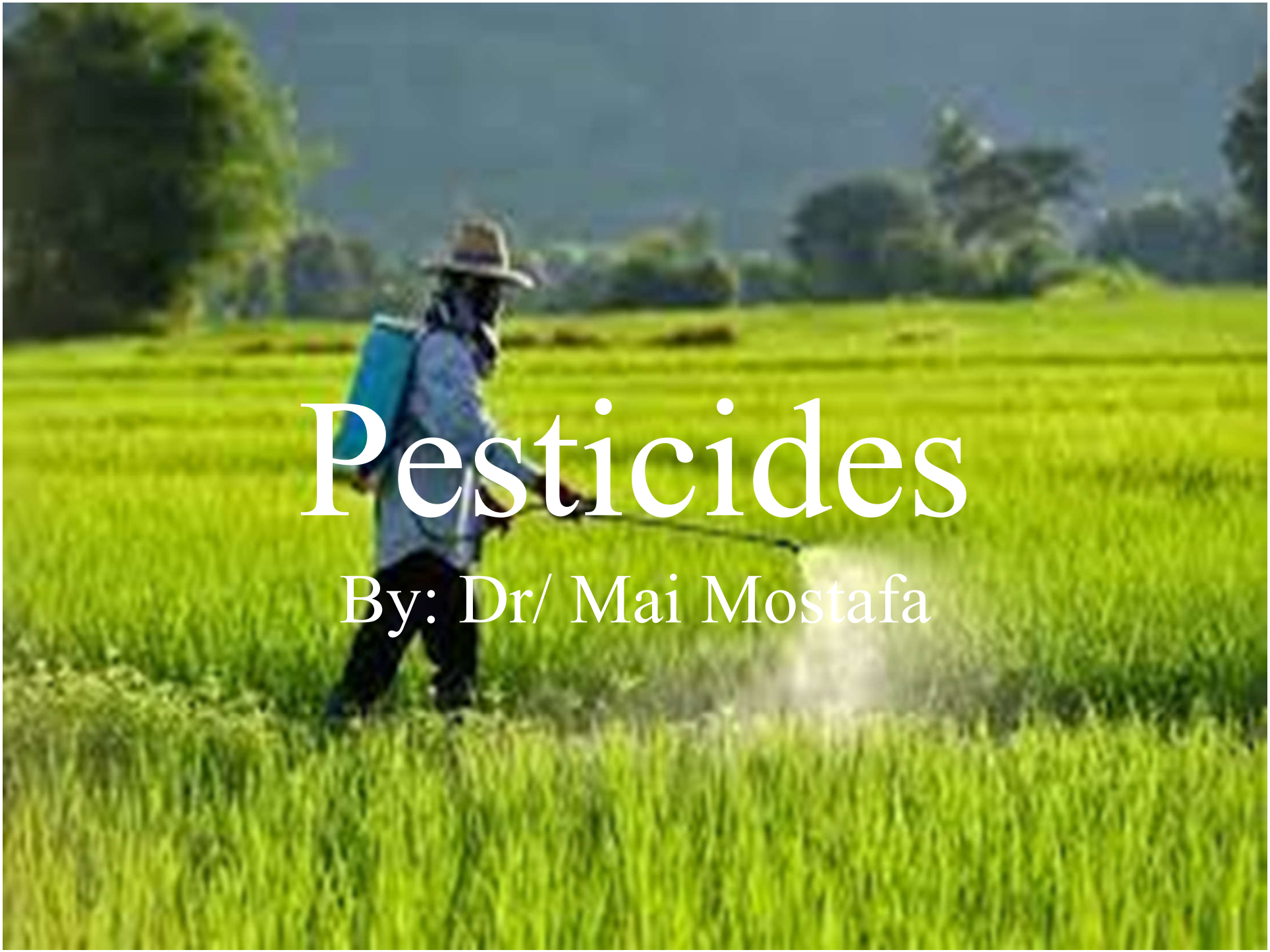

Pesticides

By: Dr/ Mai Mostafa

❖What are pesticides???

They are Any substance or a mixture of substances intended for preventing, destroying, repelling or migrating any pest .

❖Classified to:

A. Insecticides:

- Organophosphorus
- organonitrogenous (Carbamates)
- Organochlorine insecticides
- insecticides of botanical origin

B. Rodenticides

- Highly toxic thalium, yellow or white phosphorus
- Low toxicity warfarin

C. Herbicides

- Chlorophenoxy compounds
- Paraquat & Diquate

D. Fungicides

I. Organophosphorus

- **Volatile substance**
- **Has a charachtarestic garlic odor**
- **Lipophilic**
- **Used also as nerve gases in chemichal warfare**

❖Mode of poisoning:

1) **Accidental :**

   During agricultural application or manifacture and mixing these compounds

2) **suicidal:**

   as it is common, easily obtained and has a rapid action (*police El Nagda*)

3) **Homicidal:** rare by mixing with food

❖Routes of exposure:

- Oral
- Inhalation
- skin

❖Mechanism of action:

- Acetylcholine (ACh) is a neurotransmitter found at the neuromuscular junction, at preganglionic synapses in the sympathetic and parasympathetic autonomic nervous system, at postganglionic parasympathetic (muscarinic) terminals, and within the brain.

- Organophosphorous (OP) compounds inhibit cholinesterase enzymes,

    1- True (acetylcholinesterase) (AChE) (RBCs- CNS)

    2- Pseudo(plasma)

- This leads to accumulation of acetylcholine in:

1. The cholinergic nerve endings …… parasympathetic and few sympathetic nerve endings (muscarinic action)
2. The myoneural junction …. preganglionic and somatic motor nerves (nicotinic action)
3. CNS… stimulation followed by depression

- The inhibition is irreversible after 24-36 hrs without initiation of specific treatment **(aging)** .

❖Classification:

1. **Direct inhibitors:** malathion, paraxon and tetra-ethyl-pyrophosphate (TEPP)
2. **Indirect inhibitors:** parathion and chlorthion

❖Clinical presentation :

1. Acute poisoning (cholinergic crises)
2. Intermediate syndrom
3. Delayed neuropathy : 2-3 w after exposure
4. cardiotoxicity

❖ Acute poisoning:

- classical presentation starts shortly after ingestion. Corresponds in intensity to cholinestrase inhibition (50%)
- It consists of muscurinic, nicotinic and central effects.

A- Muscarinic effects of acetyl choline stimulation (DUMBLES)

- **D**iarrhea and abdominal pain
- **U**rination
- **M**iosis (pin point pupil)
- **B**radycardia, hypotension, bronchospasm and bronchorhea
- **L**acrimation
- **E**mesis
- **S**alivation, sweating,

B- Nicotinic effects (MATCH)

- Muscle fasiculations (eye lid and oral) followed by weakness and respiratory paralysis
- Adrenal medullary hyperactivity with transient hyperglycemia and mydriasis
- Tachycardia and arrhythmia
- Cramping
- Hypertension

C- Central nervous system effects include:

- Stimulation followed by depression in the form of Irritability, nervousness, ataxia, fatigue, generalized weakness.
- Depression of respiratory and circulatory centers with dyspnea, cyanosis, hypoventilation and hypotension, lethargy, impairment of memory, confusion, convulsions and coma due to inhibition of brain enzyme.

Intermediate syndrome

- It is one of the complications of organophosphorus poisoning due to inadequate oxime therapy
- It appears 1-4 days after poisoning between the crisis and onset of delayed neuropathy
- Related to the severity of poisoning not to the type of organophosphates due to prolonged inhibition of cholinestrase enzyme at motor end plate, brain & RBCs
- The patient presented by relapse of muscle weakness (cranial nerves, respiratory, neck, proximal limb muscles), decrease or absent ankle &knee reflexes and sudden resp. arrest

❖Causes of death:

1-Bronchial constriction , increased bronchial secretions , pulmonary oedema

2- paralysis of resp. ms

3- depression of resp.center

❖Management:

A- prophylactic

B- curative

A- Prophylactic measures:

- Wearing of protective clothes
- Exhaust ventillation, respirator during mixing and spraying
- Spraying by longe nozzle bottle or container with current of air
- Keep containers away from children
- Make washing facilities nearby to workers
- Workers shoud not eat, drink or smoke during the hours of work

B- Curative:

1. **ABC**
2. **Decontamination:**
   - Wash body by water
   - Remove contaminated clothes
   - Gastric lavage
3. **Antidote:** atropine & oximes.
4. **Supportive ttt:**
   - Compelet rest
   - Antibiotics
   - Return to work (cholinesterase 75%)

Atropine:

- It is the physiological antidote as it blocks the action of acetylcholine at the parasympathetic receptors
- It antagonises the muscarinic and CNS effects but not reverse the nicotinic effect.
- Given by a dose of 2-5 mg intravenous (IV) for adults every 15 min untill relief of bronchspasm and dryness of chest secretions. Do not rely on HR and pupillary size.

Oxims

- The biochemical antidote of organophosphorus.
- Oximes reactivate AChE inhibited by OP poisoning hence correcting all signs (Musc, nicot, central).
- it is essential to begin oxime therapy as early as possible before aging occure.
- The effect of oximes adjusted by measuring the level of enzymes or the disapperance of ms. Fasiculations
- Types:

  1.PAM (Pyridine Aldoxime Methiodide) pralidoxime

  2.DAM (Diacetyl Monoxime)

  3.Obidoxim (Toxogonin)

➢ Avoid:

1. CNS depressants
2. Physostigmine, neostigmine, pilocarpine as they have parasympathomimetic action.
3. Phenothiazines , theophylline have anticholinesterase activity
4. Succinyl choline ms. Relaxants

❖diagnosis:

1. **clinical picture** (cholinergic cisis and smell of OP)
2. **atrropine :** a trial of 1 mg atropine in adults (or 0.01-0.02 mg/kg in children) may be employed. The absence of signs or symptoms of anticholinergic effects following atropine challenge strongly supports the diagnosis of poisoning with OP.
3. **Cholinesterase enzyme level**

II. Carbamates

- Domestic use (Baygon), rodenticide
- Low dermal toxicity
- Rare fatal toxicity
- Reversible inhibition of cholinestrase enzymes>>> accumulation of acetyle choline>>> muscurinic and nicotinic stimulation.

➢ C/P and management similar to OP except:

- They cant penetrate BBB …….little or no CNS toxicity.
- rapid recovery…..1-2 days
- no intermediate syndrome, delayed neuropathy or cardiotoxicity
- Atropine is the only antidote
- No oximes

III. Insecticides of botanical origin

- As pyrethrum which is less toxic
- Used as household insectiside because of its rapid knock down action
- **Examples** : Raid & Ezalo tablets
- **Clinical picture:**

  GIT manifestations

  CNS stimulation Followed by depression .

  Contact dermatitis

  Asthmatic like reactions

  Sever anaphylactic reaction

  Death due to respiratory failure
- **Management:** Epinephrine

IV. Rodenticides

- **Used to control rats and mice**
- **Ex:**

1. Yellow phosphorus
2. Strychnine sulphate
3. Red squill
4. Metal phosphide:
   a) Aluminum phosphide
   b) Zinc phosphide: highly toxic, black powder, fishy odour, phosphine gas
5. Warfarine:
   - coloured pellets
   - Anticoagulant inhibit prothrombine>>> Hrg
   - Direct damage to capillary wall


ALUMINUM PHOSPHIDE TABLETS

V. Moth repellent (Naphthaline)

- Coal tar derivative, large crystaline plates with characteristic odour

- **Uses:**
  1. Moth repellent
  2. Dyes
  3. Toilet bowel deodorizer
- **Action & C/P:**
  1. Hemolysis in pt e G6PD deficiency >>> renal tubular block
  2. GIT, skin & eye irritation e contamination
  3. CNS stimulation followed by depression
- **Chronic toxicity:**
  1. A plastic anemia
  2. liver necrosis

- **Treatment:**

1.Decontamination:

  - GL
  - Activated charcoal
  - Magnesium sulphate

2.Alkaline urine e sodium bicarbonate

3.Blood transfusion

4.Hemodialysis

5.Diazepam for convulsions

6.Diet rich in CHO low in fat


Thank
You