

AGRICULTURAL POISONS

CLASSIFICATION

- **VIRTUALLY HARMLESS:** Phenoxyacetic acid plant hormones e.g. M.C.P.A, D.C.P.A, T.C.P.A
Copper oxides, oxychlorides as fungicides
Lime sulphur washes as orchard fungicides
Petroleum washes as orchard insecticides
Tar oil emulsions
- **COMPARATIVELY HARMLESS:** Sulphuric acid as weed killer, Na chlorate as mass herbicide along roads, rail tracks

CLASSIFICATION

- **MILDLY TOXIC:** Chlorinated hydrocarbon insecticides: DDT, GAMMEXANE, ALDRIN, DIELDRIN, CHLORDAN. Used to control fly, louse, tick, agri insecticides, and cattle disinfectors
- **HIGHLY TOXIC:** Arsenical compounds, (wk, oi)
Nicotine sulphates, tannates
HCN - NaCN, KCN (raticide, di)
DINITRO COMPOUNDS: (wk, insecticide,)
ORGANIC POLYPHOSPHATES: HETP, TEPP, OMPA
PARATHION, (insecticides)

CLASSIFICATION

- **VEGETABLE** :Nicotine, pyrethrins, rotenone
- **CHEMICAL INSECTICIDES**
 - **Inorganic:** phosphorus and compounds of antimony, arsenic, barium, mercury, thallium, zine and florides
 - **Organic or synthetic:** Phosphate esters

Carbamates

Chlorinated hydrocarbons

1. Indane 2. Chlorbenzene 3. BHC 4. Camphenes

Chlordane DDT Toxaphene

Aldrin phenothane Lindane
Gammaxane Strobane

ORGANOPHOSPHATES

- Highly popular insecticides because they are effective, disintegrate within days of application & do not persist in the environment
- Even minute quantities can penetrate the skin & be lethal, as evidenced by the use of organophosphate nerve gases sarin, soman, tabun, & VX in chemical weapons.

ORGANOPHOSPHORUS POISONS

- Esters of phosphoric acid 2 series of compounds
- ALKYL PHOSPHATES
- ARYL PHOSPHATES

ALKYL PHOSPHATES

1. HETP
2. TEPP (TETRON)
3. OMPA
4. DIMEFOX
5. ISOPESTOX
6. MALATHION (KILL BUG, BUGSOLINE)
7. SULFOTEPP
8. DEMETON
9. TRICLORFON

ARYL PHOSPHATES

1. PARATHION, (FOLLIDOL, KILL PHOS,EKATO)
2. PARAOXON
3. METHYL PARATHION (MEATCIDE)
4. CHLORTHION
5. DIAZINON (DIAZION,TIK 20)

- **ABSORPTION**
- **METABOLISM**
- **EXCRETION**
- Peculiar smell due to addition of solvent usually **AROMAX** which has kerosene like smell.

ORGANOPHOSPHATES POISONING.

- The organophosphates irreversibly inhibit acetylcholinesterase, resulting in an overabundance of acetylcholine at synapses & the myoneural junction.
- The acetylcholine initially excites & then paralyzes the CNS, the parasympathetic nerve endings & the sweat glands (muscarinic effects), somatic nerves & ganglionic synapses of autonomic ganglia (nicotinic effects).
- Initial symptoms resemble a flulike syndrome with abdominal pain, vomiting, headache, dizziness.

ORGANOPHOSPHATES POISONING.

- The full-blown picture generally develops by 24 hours ,includes coma, convulsions, confusion, or psychosis; fasciculation , weakness or paralysis; dyspnea, cyanosis, pulmonary edema; sometimes pancreatitis.
- Torsades de pointes VF has also been described.

● **SIGNS AND SYMPTOMS**

- Occur when Achestrase levels drop to 30% of its normal activity
- Ocular exposure --- persistent miosis
- Onset of systemic symptoms varies with different routes of absorption
- Involuntary Ms + secretory glands are affected first followed by voluntary Ms then brain functions
- Symptoms may begin within 5 min to 2 hrs
- And are max in 24 hrs
- Respi and G.I.T symptoms are more marked depending on the route of entry

SIGNS AND SYMPTOMS

1. MUSCARINIC MANIFESTATIONS

Easily remembered as S

Salivation

L

Lacrimation

U

Urination

D

Defaecation

G

G I Distress

E

Emesis

SIGNS AND SYMPTOMS

- **DUMBELS**

- D-diaphoresis, diarrhea, decreased BP
- U-urination
- M-miosis
- B-bronchorrhea, bronchospasm, brady
- E-emesis, excitation of skeletal muscle
- L-lacrimation
- S-salivation, seizures

SIGNS AND SYMPTOMS

1. MUSCARINIC SYMPTOMS

1. Respiratory System: Bronchoconstriction, increased bronchial secretions, dyspnoea, cyanosis, pulmonary oedema
2. G.I.T : A/N/V cramps, diarrhoea, fecal incontinence, tenesmus
3. Sweat glands:
4. Salivary glands:
5. Lacrimal glands:
6. C.V.S: Bradycardia , hypotension
7. Pupils:
8. Ciliary Body: Blurred vision
9. Bladder : Urinary incontinence

SIGNS AND SYMPTOMS

2. NICOTINIC MANIFESTATIONS

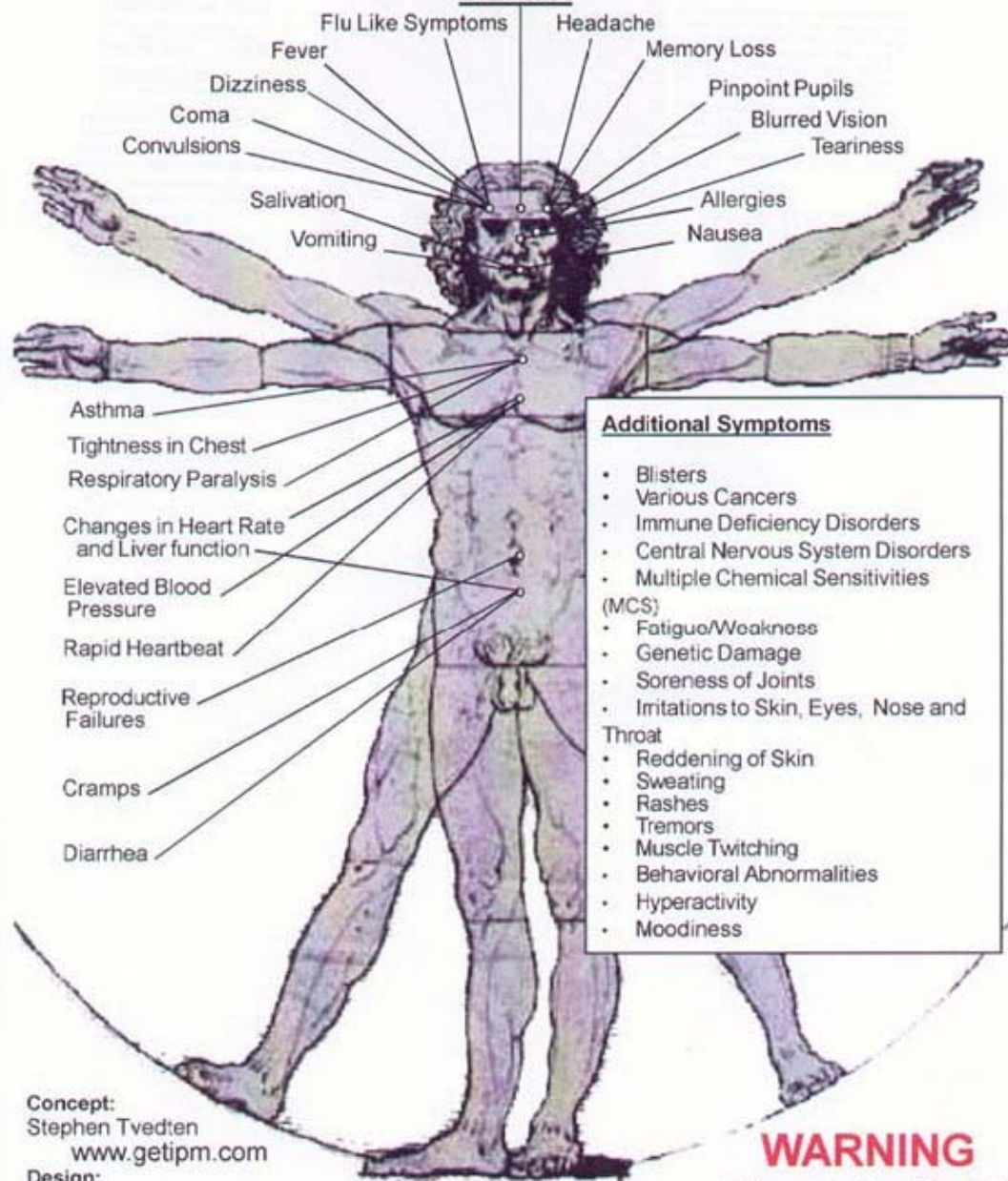
- **STRIATED MUSCLE:** Initial stimulus results in contraction, later there is paralysis due to persistent depolarisation
- **Ms weakness** is due to accumulation of Ach which causes fasciculation, cramps, weakness , areflexia
- **SYMPATHETIC GANGLIA:** HT, Tachycardia, pallor, mydriasis

SIGNS AND SYMPTOMS

3. CNS MANIFESTATIONS

- Restlessness, emotional lability, headache, tremor, drowsiness, confusion, slurred speech, ataxia, generalised weakness, coma, convulsion, depression of respi and CVS centres

DEATH



Concept:
Stephen Tvedten
www.getipm.com
Design:
Linda L. Jensen-Pascarella
www.safe2use.com

©1999, 2000 Safe2Use

WARNING
When Used as Directed
Pesticides Kill

- **Mild poisoning** : S/S are malaise , nausea, fatigue, minimal Ms weakness, cramping without diarrhoea
- **Moderate poisoning**: SLUDGE and / or tremors, weakness, fasciculations, confusion, lethargy, anxiety
- **Severe poisoning**: SLUDGE and respiratory insufficiency, weakness, fasciculations, coma, paralysis, seizure, autonomic dysfunction.
- Porphyrinemia resulting in **CHROMOLACHRYORRHOEA** (shedding of red tears) due to accumulation of porphyrin in the lacrimal glands is seen very rarely

- **INTERMEDIATE SYNDROME:** After 1 to 4 days Ms weakness and paralysis char. By cranial nerve palsies, weakness of neck flexors, and proximal limb Ms, and acute respi paralysis are seen due to prolonged cholinesterase inhibition and Ms necrosis. It does not respond to oximes or atropine

Delayed Sequelae:

Delayed peripheral neuropathy in 1 -- 5 weeks

- Parathion, malathion, trichlorfon
- Begins with parasthesias and pain or cramps in calf Ms followed by ataxia, weakness and toe drop.
- It rapidly progresses to a flaccid paresis which can ascend similar to Guillian Barre syndrome.
- Reflexes are diminished
- The disease may progress for 2-3 months and Ms wasting occurs

CHRONIC POISONING

- Occupational hazard
- Main features are Ms cramps, ataxia, generalised weakness, gait disturbances, confusion, irritability, anxiety, etc.
- FATAL DOSE
- TEPP 50 mg i.m or 100 mg orally
- OMPA 80 mg i.m or 175 mg orally
- Parathion 80 mg i.m or 175 mg orally
- HETP 60 mg i.m or 375 mg orally
- Malathion and Diazinon 1gm orally

FATAL PERIOD

- Usually within 24 hrs in untreated cases
- And within 10 days in cases where t/t not successful
- Cause of death:
 1. Paralysis of respi Ms
 2. Respiratory arrest due to failure of respiratory centre
 3. Intense bronchoconstriction
 4. Ventricular arrhythmias (Late)

DIAGNOSIS

- 5 ml of heparinised blood for cholinestrase determination
- The cholinestrase activity of blood and plasma fall by 22 to 88%.
- The avg normal values are 77 to 142 in the RBC's and 41 to 142 in the plasma
- Diagnosis may be confirmed by giving 2 mg of atropine.
- A 25% reduction in red blood cell cholinesterase confirms organophosphate poisoning.

TREATMENT

- Emergency management includes decontamination of the skin, & removal of clothes;
- Establishing an airway & ensuring proper ventilatory support , cardiac monitoring;
- Gastric Lavage with 1:5000 KMnO₄
- Activated Charcoal one gm/kg
- Atropine sulphate arrests the muscarinic and CNS symptoms . It has no effects on the nicotinic symptoms
- 2-4 mg of atropine is given i.v as a test dose
- If no effect this dose is doubled every 5-10 min until muscarinic symptoms are relieved
- An avg patient requires 40 mg per day but upto 1000mg /day have been used

TREATMENT

Atropine should be given as a physiologic antidote to reverse the muscarinic effects & to dry the excessive pulmonary secretions seen in patients with respiratory distress.

- Atropine use requires cardiac monitoring & proper oxygenation.
- Specific cholinesterase activators like diacetyl monoxime DAM, or 2-pyridine aldoxime methiodide PAM Pralidoxime is the **treatment of choice** for organophosphate poisoning & should be begun on clinical grounds before return of any blood studies.
- To be effective, pralidoxime must be given in the first 48 hours before irreversible binding of acetylcholinesterase occurs.

- Dose is 1-2 gm i.v either as a 5% sol. Given over 5 min or in 150 ml of saline and infused over half An hour
- Can be repeated if Ms weakness not relieved
- Repeat in 6-12 hrs for 24 to 48 hrs
- Max dose of 12 gms should not exceed in 24 hrs
- Pralidoxime may obviate the need for high-dose atropine therapy & reduce the incidence of late-onset paralysis.
- Neither therapies exclude the use of the other.
- Obidoxime chloride is more potent but toxicity greater
- HI-6 and H10-7 appear to have activity against all known OP
- Convulsions controlled by Diazepam
- Rest symptomatic

PROPHYLAXIS

1. Protective clothing
2. Washing of hands and face
3. Less than 2 hrs of spray
4. Proper instructions for workers
5. No smoking, chewing, drinking, eating in area of spray
6. Washing of equipments after spraying
7. Stop spraying immediately if rash, feeling sick etc.

- **POST MORTEM APPEARANCES**

- Signs of asphyxia

- Chronic poisoning

- Poisoning:

CARBAMATES

- Derivatives of carbonic acid
- E.g are aldicarb (temik), aminocarb (metacil),
 aprocarb (baygon), carbaryl (sevin)
 carbofuran (furaxdan)
 Carbaryl, carbofuran, propoxur, methomyl are
 highly toxic

S/S: begin within 15 minutes to 2 hrs.

Carbamates differ toxologically from OP

1. Spontaneously hydrolyse from Acheesterase enzymatic site
within 24-48 hrs

CARBAMATES

- 2. Do not penetrate the CNS hence CNS toxicity is limited
- Rest s/s same as OP
- T/t : ATROPINE is the specific antidote
- Pralidoxime may diminish the severity of the symptoms and help prevent some morbidity
- It improves respi functions and patient well being

ORGANOCHLORINES

- CAN BE DIVIDED INTO 4 CATEGORIES

1. Indane 2. Chlorbenzene 3. BHC 4. Camphenes

Chlordane

Lindane

Toxaphene

Aldrin DDT phenothane Gammexane Strobane

- Absorption

- FATAL DOSE: DDT 30 gms

Gammexane 15 gms

Lindane 15 gms

Chlordane 30 gms

ENDRIN

- Cyclodine insecticide, stereoisomer of dieldrin
- Soluble in aromatic hydrocarbons, not soluble in water
- Bitter taste, also called “plant penicillin”
- Endrin-We-16, Endox-DB-50, Endtox-EC-20, Endrex

S/S: Begin within 1-6 hrs

Chronic poisoning: Main features are Ms cramps, ataxia, generalised weakness, gait disturbances, confusion, irritability, anxiety, oligospermia, increased tendency to leukemias, purpura, aplastic anaemia and liver cancer etc.

FATAL DOSE: 5-6 gms

FATAL PERIOD: One to several hrs

- PM appearance: Body smells of kerosene, signs of asphyxia present
- Endrin resists putrification
- T/t : Cholestyramine is a non absorbable bile acid binding anion exchange resin, which increases the fecal excretion of organochlorines
- No specific antidote

PARAQUAT

- Bipyridium compound, used as herbicide and weed killer
- Gramoxone, Weedol
- Death due to inhalation rare, but by ingestion is frequent
- M.O.A : It undergoes NADPH dependent reduction to form the free radical which reacts with Oxygen to produce superoxide free radicals
- Absorption and excretion: Highest conc. In kidneys and lungs followed by muscles from where redistribution back to circulation takes place

- > than 90% of absorbed paraquat is excreted unchanged in urine in first 24 hrs
- **FATAL DOSE:** 3-5 gms
- **FATAL PERIOD:** 2-7 days
- S/S: Ingestion of > 50mg/kg kills within 3 days
- **Local:** Irritation and inflammation of skin, nails, cornea, conjunctiva
- G.I.T: Oropharyngeal ulceration and corrosion, N/V/D, Hematemesis, dysphagia, aphonia, perforation mediastinitis, pneumothorax
- Kidneys: Oliguria, ATN, non oliguric renal failure
- Lungs: Cough, haemoptysis, dyspnoea due to pulm oedema,

- Pancreas:
- Liver: Centrilobular hepatic necrosis, cholestasis
- C.V.S: Hypovolemia, shock, arrhythmia
- C.N.S: Convulsion, coma
- Adrenals: Insufficiency due to necrosis
- Bone marrow: PMN Leucocytosis, late anemia
- **CAUSE OF DEATH:** Multi organ failure

Corrosive effects

Mediastinitis, perforation

TREATMENT

1. GL
2. One litre of 15-30% of aq. Suspension of Fullers earth or 7% bentonite are given to ADSORB paraquat, followed by 200ml of 20% mannitol
3. AC
4. Hemodialysis and Hemoperfusion useful if done within 12 hrs
5. Avoid oxygen therapy
6. Analgesics

PM Appearances:

ALUMINIUM PHOSPHIDE (ALP)

- Solid fumigant pesticide, insecticide, rodenticide
- Celphos, Alphos, Quickphos, Phostoxin, Phosphotex each weighing 3 gms and have the capacity to liberate 1gm of PHOSPHINE (PH_3).
- On coming in contact with moisture ALP liberates PH_3
- PH_3 is a systemic poison
- HCl accelerates the liberation of PH_3
- ALP has garlicky odour

Absorption and excretion: Rapidly absorbed by simple diffusion

ALUMINIUM PHOSPHIDE (ALP)

- After ingestion , some ALP is absorbed and metabolised in liver, where PH_3 is slowly released accounting for its prolonged symptoms
- PH_3 is oxidised slowly to oxyacids and excreted in the urine as hypophosphite. Also excreted unchanged from lungs
- Action: PH_3 inhibits respi chain nzymes and has cytotoxic action
- **FATAL DOSE:** 1-3 tablets
- **FATAL PERIOD:** 1 hr-4 days
- **S/S:** Inhalation: Irritation and inflammation of mucous membrane, N/V/D/Headache, dizziness, easy fatigue, tightness in the chest.

ALUMINIUM PHOSPHIDE (ALP)

- **Moderate toxicity** produces ataxia, numbness, tremor, diplopia, parasthesia, jaundice, Mus weakness, incoordination and paralysis
- Conc. in air higher than 0.3 ppm causes severe illness
- **Severe toxicity** produces ARDS, Cardiac arrhythmias, CHF, Pulm oedema, convulsion, coma
- **Ingestion:** G.I.T: N/V/D, retrosternal pain
 - Kidneys: Renal failure
 - Lungs: Cough, haemoptysis, dyspnoea due to pulm oedema, respiratory failure

- Pancreas:
- **Liver:** Jaundice, hepatitis, hepatomegaly
- **C.V.S:** Hypovolemia, shock, arrhythmia, myocarditis, pericarditis, Acute CHF
- **C.N.S:** Headache, dizziness, altered mental state, restlessness, Convulsion, coma, Acute hypoxic encephalopathy
- Rarely Ms wasting and tenderness and bleeding diathesis
- **CAUSE OF DEATH:** Cardiogenic shock
- Complications: Pericarditis, Acute CHF, massive G.I bleed, ARDS

PM Appearances:

- Garlic like odour
- Blood stained froth
- Congested mucous memb
- Rest of organs congested
- Centrizonal haemorrhagic necrosis in liver
- Histopathology:
- Chemical test: Gastric aspirate 5ml and 15 ml of water in a flask, cover it with filter paper impregnated with Silver nitrate, heat the flask at 50 degrees for 15 min the filter paper turns black

Filter paper impregnated with Silver nitrate used in form of mask through which patient breaths for 5-10 min it turns black

TREATMENT

1. GL
2. AC
3. ANTACIDS
4. Liquid paraffin
5. No specific antidote
6. MgSO_4
7. Symptomatic