
بِسْمِ ٱللَّهِ ٱلرَّحْمَٰنِ ٱلرَّحِيمِ

ORGANOPHOSPHORUS POISONING

organophosphorus

- Pathophysiology
- Clinical picture
- complications
- Investigation
- treatment

Pathophysiology:-

- Inhibition of acetylcholinesterase enzyme
- Accumulation of acetylcholine
- Stimulate muscarinic and nicotinic receptors as well as CNS.
- Irreversible after 24-36h.

Muscarinic effects:-

- Diarrhea
- Urination
- Miosis
- Bradycardia , hypotension, bronchospasm
- Vomiting
- Lacrimation
- Salivation,sweating

Nicotinic effects:-

- Muscle fasciculation
- Hyperglycaemia
- Tachcardia,dysrhythmia
- Cramping of skeletal muscles
- hypertension

Central effects:-

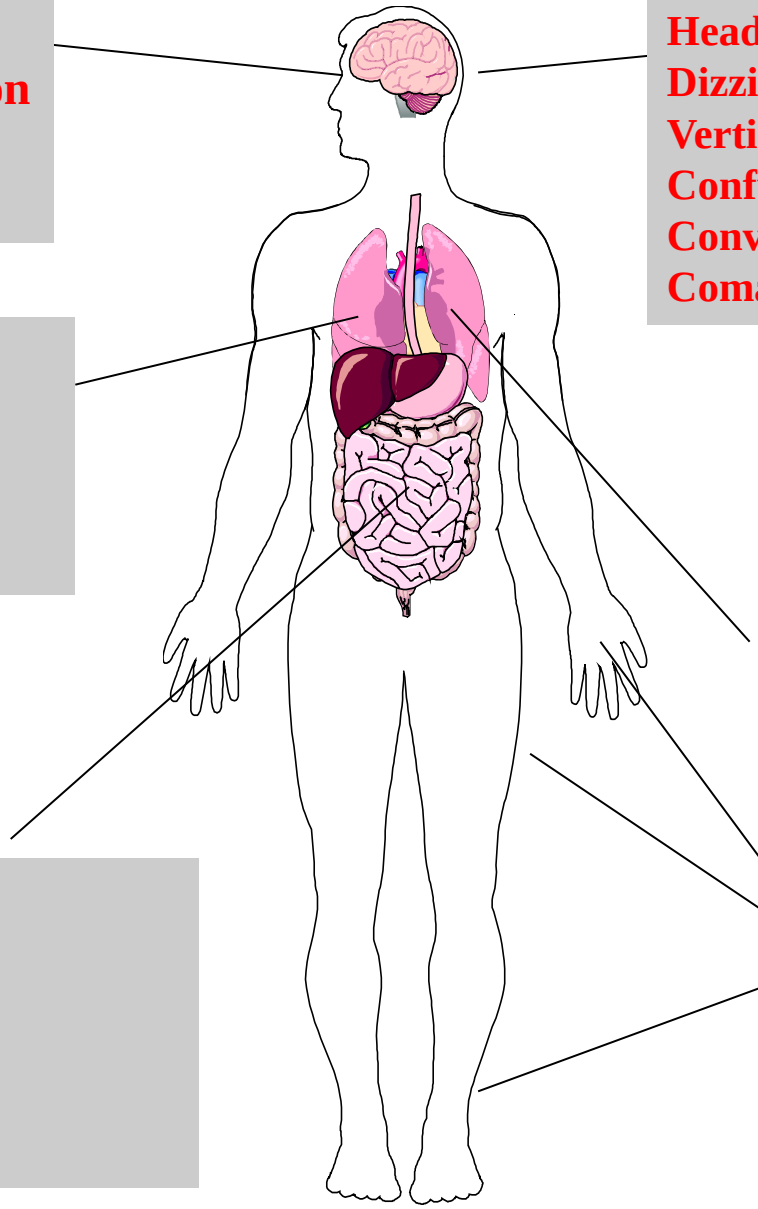
- Vertigo
- Tremors
- Convulsions
- Confusion
- coma

Manifestations of Organophosphate Poisoning

eyes
Pupil Constriction
Blurred Vision
Lacrimation

CNS
Headache
Dizziness
Vertigo
Confusion
Convulsion
Coma

Respiratory System
Bronchospasm
Bronchial Secretion
Pulmonary Edema
Wheezing



Cardiovascular System
Tachycardia
hypertension

Gastrointestinal Tract
Salivation
Nausea
Abdominal Pain
Vomiting
Diarrhea

Musculature
Weakness
Tremor
Fasciculations
Twitching
Cramps
Increased Sweating

Complications:-

- Intermediate syndrome
- Delayed neuropathy
- Cardiotoxicity

Investigation:-

- Arterial blood gas, glucose
- ECG
- CHEST X-RAY
- Assessment of true and pseudo cholinesterase enzymes.

	TURE CHOLINESTERASE	pseudo cholinesterase
advantages	Better reflection of synaptic inhibition	Easier to assay Faster decline
site	Red blood cells brain	Plasma –liver, Pancreases -heart
regeneration	1% day	25%-30%in first 7-10 days
normalization	35-100 day	28-42 day
False depression	Pernicious anemia Antimalarial Anti depressant	Liver dysfunction Malnutrition Drugs(succinylcholine,codeine,morphine)

Treatment:

- Emergency treatment
- Decontamination
- Antidotes
- Symptomatic treatment
- Treatment of complications

Decontamination:-

- wash the body with cold then hot water
- If there is smell in hair cut it to avoid continuous absorption
- Gastric lavage with cuffed ETT
- AC

Specific treatment:-

- Atropine
- Competitive antagonist of acetylcholine at muscarinic receptors
- 2-5mg IV
- Maintenance dose for 1-2 days to avoid possible relapses of cholinergic crisis.

Cholinesterase reactivators:-

- Oximes reactivate the inhibited cholinesterase
- Given at first 48 h
- Pralidoxime (PAM)
- **Reactivates The Phosphorylated AchE By Binding To The OP Molecule.**
- 1-2mg IV as loading dose followed by half this dose every 6-8 h for 2 days

Obidoxime (toxogonin)

- Cross BBB
- Antagonise muscarinic ,nicotinic and central effects
- 250 mg slowly IV
- 750 mg /24h

Benzodiazepines

- 5-15 Mg IV Q5-10 Min
- Routinely Used In OP Poisoning For Treatment Of Agitation And Seizures

New treatment:-

- Sodium bicarbonate
- Magnesium sulphate
- Clonidine
- Beta adrenergic agoinsts

Sodium bicarbonate:-

- Enhance oragnophosphorus clearance
- Improve efficacy of oximes

Magnesium sulphate

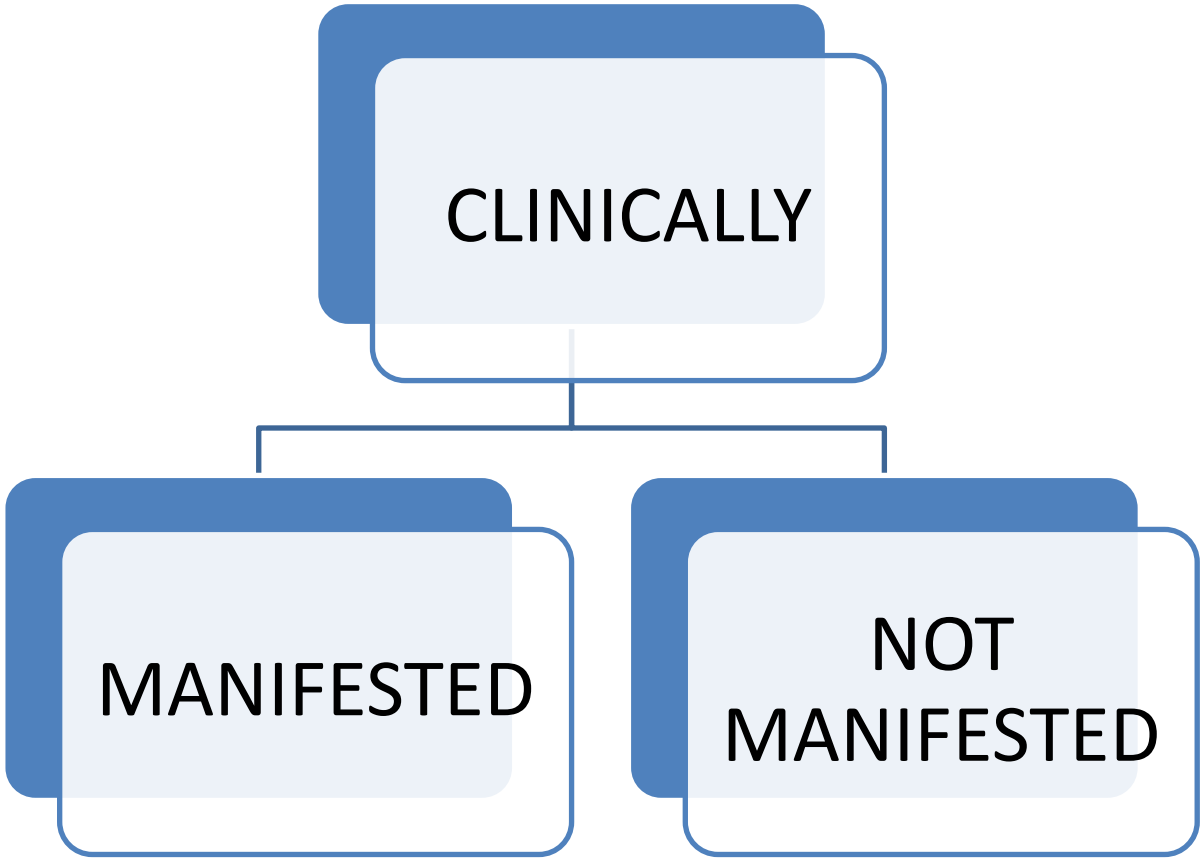
- Reduce synaptic acetylcholine release by blocking calcium channels
- Reducing cholinergic stimulation
- Reduce the risk of ventricular tachycardia

clonidine

- Reduce synaptic acetylcholine release
- Inhibit muscarinic effects of organophosphorus.

beta-adrenergic agonists salbutamol or albutrol

- Treating bronchospasm
- Enhance effect of atropine in decreasing lung fluid (bronchorrhea)
- Enhance removal of fluid in lung


CLINICALLY
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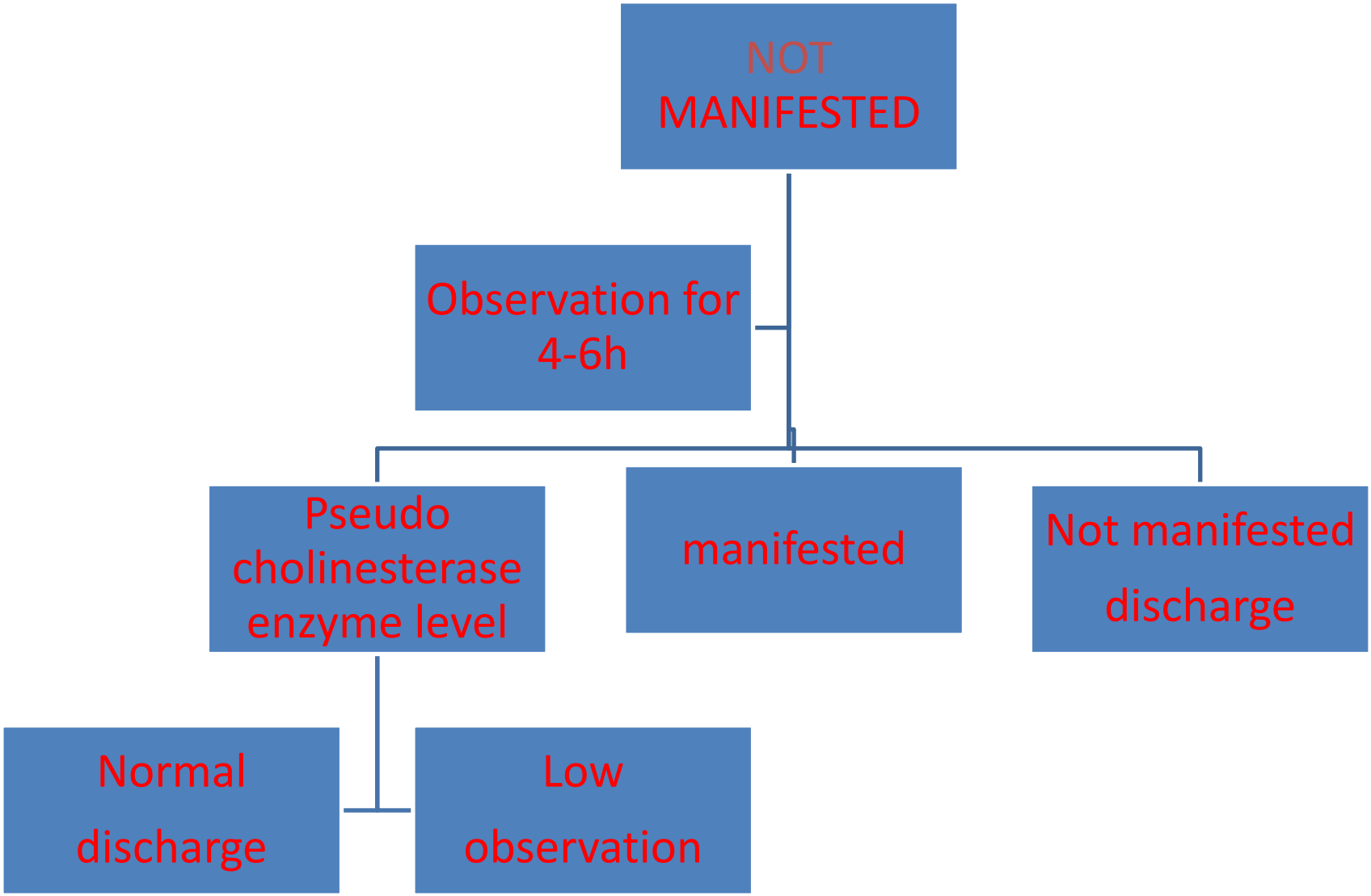
NOT MANIFESTED
Observation for 4-6h
Pseudo cholinesterase enzyme level
manifested
Not manifested discharge
Normal discharge
Low observation

THANKS