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ORGANOPHOSPHATES and CARBAMATES

GENERAL INFORMATION

This protocol refers to pesticides and military nerve agents (Sarin, Soman, Tabujn, VX).

Symptoms usually occur with 1-2 hours of exposure but may be delayed up to 6-8 hours (skin exposure) or with those that require metabolism to active toxic metabolites (prothiofos, chlorpyrifos) (up to 12 hours).

Hydrocarbons are often the vehicle for organophosphate preparations. Aspiration of the hydrocarbon may be a complication of the clinical presentation. Refer to Hydrocarbons for further management.

CLINICAL EFFECTS

CHOLINERGIC TOXIDROME:

Muscarinic Signs and Symptoms	**Nicotinic Signs and Symptoms**	**Central Signs and Symptoms**
"DUMBELS"	**"MATCH"**	
Defecation	**M**uscle weakness/fasciculations	Agitation
Urination	**A**drenergic stimulation	Seizures
Miosis (small pupils)	**T**achycardia	Coma
Bronchospasm/bronchorrhea/ bradycardia	**C**ramping of skeletal muscles	
Emesis	**H**ypertension	
Lacrimation	Mydriasis is possible.	
Salivation	Respiratory depression also occurs.	

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TREATMENT

GENERAL CONSIDERATIONS

- The risk of secondary poisoning of staff from a patient with pesticide exposure is low when basic precautions are followed.
- Resuscitation and treatment should occur in a well-ventilated area with regular rotation of staff (every 30 minutes).
- All staff with direct patient contact should use universal precautions (gowns, gloves, eye protection, and masks).
- External decontamination of the patient should not delay timely resuscitation and medical attention.
- Staff with inadvertent direct contact to a patient's body fluids should wash the affected area immediately and thoroughly.
- The odour associated with pesticide-exposed patients is most often related to the hydrocarbon vehicle and not the pesticide compound. Prolonged exposure to these odours may result in self-limited symptoms that can be reduced by working in a well-ventilated area and rotating staff.

DERMAL EXPOSURE

- Decontamination should ideally occur at a location outside the hospital.
- Patient clothing should be removed, bagged, and discarded.
- Wash the skin and hair with soap and water.
- Proceed with oral exposure treatment, omitting GI decontamination step.

ORAL EXPOSURE

- Maintain a patent airway, assess respiratory muscle function and aid ventilation if necessary.
- Single dose activated charcoal should be given within 2 hours of ingestion.
- Atropine
    - Atropine treatment should be the priority for both organophosphate and carbamate exposures.
    - Reverses muscarinic symptoms only.
    - See dosing below.

- Pralidoxime (2-PAM)
    - Only required for organophosphate exposures, not for carbamate pesticide exposures.
    - Treatment for nicotinic or central symptoms.
    - Any organophosphate exposed patient requiring atropine for muscarinic symptoms should receive at least one dose of 2-PAM.
    - If exposure is unknown (i.e. organophosphate vs carbamate), err on the side of giving atropine plus 2-PAM.
    - Most effective when given in first 24 hours, but may be given later.
    - See dosing below.

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ATROPINE

Indications for atropine following an organophosphate or carbamate pesticide exposure include any of these signs of a cholinergic toxidrome:

1. Miosis/small pin-point pupils
2. Excessive sweating
3. Respiratory distress: poor air entry, bronchorrhea, bronchospasm/wheeze
4. Bradycardia (normal or fast heart rates are also common)
5. Hypotension

If none of these signs present, there is no need for atropine. Continue to monitor patient for these signs.

If uncertain about the type of ingestion, give 1mg (adult) (0.01 mg/kg – pediatric) atropine IV/IM/SQ/ETT test dose. If heart rate increases more than 20-25 beats/min and skin gets flushed, significant cholinergic toxicity unlikely and no further atropine needed.

ATROPINE DOSING:

Start with 2 mg (adult) (0.02 mg/kg – pediatric) atropine IV/IM/SQ/ETT bolus. Then double the dose of atropine every 3-5 mins until improvement in all 4 target end-points of atropinization:

1. chest clear on auscultation, no wheeze
2. heart rate > 80 beats/min
3. systolic blood pressure > 80 mmHg
4. dry axillae

Once atropinized, start an infusion of atropine at 10-20% of total atropine dose required to atropinize the patient per hour (maximum 3 mg/hour). Infusion rate may need to be titrated up or down depending on signs of over- or under- atropinization.

COMPLICATIONS OF ATROPINE TREATMENT:

Agitation, confusion, delerium, hallucinations
Urinary retention
Bowel ileus
Hyperthermia
Excess tachycardia, may lead to myocardial infarctions in patients with pre-existing heart disease

Tachycardia alone is **not a contraindication for atropine if other signs of cholinergic toxicity are present. Tachycardia due to hypoxia will improve with atropine once chest is clear and oxygenation improves.

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PRALIDOXIME (2-PAM)

Not recommended for carbamate poisoning.

PRALIDOXIME DOSING:

For organophosphate poisoning give:

Pralidoxime 30 mg/kg IV over 20 min.
Followed by 8 mg/kg/hr continuous IV infusion until clinical recovery.

Titrate according to respiratory effort and muscle strength. May need to continue infusion for days.

COMPLICATIONS OF PRALIDOXIME:

Tachycardia, laryngospasm, muscle rigidity, vomiting and hypertension have been attributed to rapid administration of pralidoxime.

SPECIAL CONSIDERATIONS

- Plasma cholinesterase activity can be measured but has no place in the acute management of the intoxicated patient.
- "Intermediate syndrome"
  - Respiratory and skeletal muscle weakness beginning 1-4 days after initial recovery from cholinergic poisoning. Signs include weak neck flexion, respiratory distress, cranial nerve palsies, and proximal muscle weakness. May last up to 15 days.
  - Treatment is respiratory support. Pralidoxime and atropine are not effective.
- Delayed neuropathy
  - A distal sensory-motor polyneuropathy may occur 6-21 days after exposure. Symptoms may be prolonged or permanent. No effective treatment is available.

REFERENCES

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