# OP poisoning in Children a review

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## ABSTRACT

Poisoning is a relatively important health problem among children. Each year around the world approximately two million children under the age of five are poisoned. Children are adventurous and inquisitive, hence the high incidence of poisoning being recorded during their developmental period. The majority of poisoning cases are involuntary and inadvertent. Children may be victim of different types f poisoning and among them organophosphorous compound poisoning is in our day to day practice.

Organophosphorous compounds which are available in different forms and used as agricultural and household insecticides and in the treatment of animal ectoparasites and human lice infestation. These substances are absorbed via the lungs, skin and the alimentary tract. Food including stored grains contaminated by a spray of these insecticides had been known to cause severe poisoning.

This review article will remind the history of OP poisoning in short and will discuss about the classification of poisoning according to the toxicity of the compounds. Further it will discuss about the clinical features of this poisoning and lastly briefly talk about its management.

**Keyword:** children, insecticides, organophosphorous

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Organophosphorus compounds are chemical agents in wide-spread use throughout the world, mainly in agriculture. Organophosphates were first discovered more than 150 years ago; however, their widespread use began in Germany in the 1920s, when these compounds were first synthesized as insecticides and chemical warfare agents. Interest in the effects of these compounds on humans has increased in recent years due to their potential use as weapons of mass destruction.[1]

Organophosphates and carbamates are the most frequently used insecticides worldwide. These compounds cause 80% of the reported toxic exposures to insecticides. Organophosphates produce a clinical syndrome that can be effectively treated if recognized early. The typically described clinical syndrome in adults often does not occur in young children.[2-5]

Most symptoms appear within 12-24 hours of exposure. Exposure can occur by means of ingestion, dermal exposure, or inhalation. Children often ingest home pesticides they find in unmarked or poorly stored containers. Children can also be exposed when playing in areas recently treated with organophosphate compounds. A history of possible exposure combined with physical signs and symptoms consistent with exposure often lead to diagnosis. Many organophosphates can irritate the skin and mucous membranes. Some have a characteristic odor, smell.

There are no rules and regulations governing the purchase of these products, and they are therefore readily available "over the counter", despite them being a major cause of morbidity and mortality. Exposure to organophosphates in an attempt to commit suicide is a key problem, particularly in the developing countries, and is a more common cause of poisoning than the chronic exposure experienced by farmers or sprayers in contact with pesticides. Estimates from the WHO indicate that each year, 1 million accidental poisonings and 2 million suicide attempts involving pesticides occur worldwide. Intoxication occurs following absorption through the skin, ingestion via the GI tract or inhalation through the respiratory tract.6

**Classification:**

There are more than a hundred organophosphorus compounds in common use. These are classified according to their toxicity and clinical 1 use:

1. Highly toxic organophosphates: (e.g. tetra-ethyl pyrophosphates, parathion). These are mainly used as agricultural insecticides.
2. Intermediately toxic organophosphates: (e.g. coumaphos, clorpyrifos, trichlorfon). These are used as animal insecticides.
3. Low toxicity: (e.g. diazinon, malathion, dichlorvos). These are used for household application and as field sprays.6

Organophosphates form an initially reversible bond with the enzyme cholinesterase. The organophosphate-cholinesterase bond can spontaneously degrade, reactivating the enzyme, or can undergo a process called aging. The process of aging results in irreversible enzyme inactivation.

Cholinesterase is found in 2 forms: an RBC form, which is known as true cholinesterase, and a plasma form, which is known as pseudo cholinesterase. Cholinesterase rapidly hydrolyzes the neurotransmitter acetylcholine into inactive fragments. Acetylcholine is found in sympathetic and parasympathetic ganglia and in the terminal nerve endings of postganglionic parasympathetic nerves at the motor endplates of nerves in the skeletal muscle. Inactivation of the enzyme allows acetylcholine to accumulate at the synapse, leading to overstimulation and disruption of nerve impulses. Skeletal-muscle depolarization and fasciculation occur secondary to nicotinic stimulation at the motor endplate.

Muscarinic effects occur at the postganglionic parasympathetic synapses, causing smooth-muscle contractions in various organs including the GI tract, bladder, and secretory glands. Conduction can be delayed in the sinus and atrioventricular (AV) nodes. Dysrhythmias are frequently reported; these typically include bradycardia, though tachycardia can also occur.

Predominant symptoms and signs vary according to the age of the affected person. Children, particularly young children, present with altered levels of consciousness rather than the classic DUMBELS signs that are most commonly observed in adults.[7]

Acetylcholine receptors are widely dispersed throughout the CNS. The activation of these receptors causes a wide range of effects, including CNS stimulation, seizures, confusion, ataxia, coma, and respiratory or cardiovascular depression.

Organophosphates are generally highly lipid soluble and are well absorbed from the skin, mucous membranes, conjunctiva, GI system, and respiratory system.[7]

Zwiener and Ginsburg (1988) retrospectively examined 37 patients aged 1 month to 11 years who had been exposed to insecticides. The most common signs were miosis, excessive salivation, muscle weakness, and lethargy. Approximately 49% of these children presented with tachycardia.[8]

Lifshitz et al (1999) retrospectively examined 36 children aged 2-8 years who were exposed to organophosphates or carbamates in Israel. The authors observed a decreased level of consciousness, including coma, stupor, and hypotonicity in all children.[9]

Lima and Reis (1995) reported carbamate poisoning in Rio de Janeiro. Symptoms included salivation, lacrimation, urination, defecation, GI distress, and emesis (SLUDGE) and were more commonly observed in adults than in children.[10]

Sofer et al (1989) retrospectively examined 25 patients aged 3 months to 7 years with carbamate or organophosphates poisoning in Israel.The most common presenting symptoms were CNS depression, stupor, coma, and flaccidity. The classic SLUDGE symptoms were more likely to be absent in these children.[11]

The classic muscarinic and nicotinic signs of intoxication including increased secretions, bradycardia, fasciculations, and miosis were less common in our patient population.

Toxicity due to various poisons, such as carbamates, phosgene, paraquat and nerve agents, can cause symptoms similar to those of organophosphates.

In young children, suspect organophosphate poisoning if they have any illness that depresses the level of consciousness.

Anticholinergic agents are important for controlling the life-threatening effects of organophosphate exposure. Initiate atropine therapy early to control secretions, bronchoconstriction, bronchospasm, and GI toxicity. 2-PAM is an oxime that reactivates cholinesterase, restoring respiratory and skeletal muscle strength. 2-PAM does not cross the blood-brain barrier; hence, the central effects are not reversed.

Health care providers must avoid contaminating themselves while handling patients. Use personal protective equipment such as gloves and gowns, when decontaminating patients because hydrocarbons can penetrate nonpolar substances such as latex and vinyl. Use charcoal cartridge masks for respiratory protection when decontaminating patients who are significantly contaminated.

Airway control and adequate oxygenation are paramount in organophosphate (OP) poisonings. Intubation may be necessary in cases of respiratory distress due to laryngospasm, bronchospasm, bronchorrhea, or seizures. Immediate aggressive use of atropine may eliminate the need for intubation.

**Attention on following points**

- Remove all clothing and gently cleanse patients suspected of organophosphate exposure with soap and water
- Continuous cardiac monitoring and pulse oximetry should be established; an ECG should be performed
- Patients with trauma or blast injury should be treated according to standard advanced trauma life support (ATLS) protocol. Patient decontamination should always be considered to prevent medical personnel poisoning.

The mainstays of medical therapy in organophosphate (OP) poisoning include atropine, pralidoxime (2-PAM), and benzodiazepines (eg, diazepam.

Because large amounts of atropine may be required for patients with organophosphate poisoning, reconstitution of powdered atropine is a viable option, especially in mass-casualty settings. Recently, Rajpal et al demonstrated the clinical safety and efficacy of sublingual atropine to healthy volunteers. This may offer another route of administration for the OP poisoned patient, especially in a mass-casualty scenario

Intravenous glycopyrrolate or diphenhydramine may provide an alternative centrally acting anticholinergic agent used to treat muscarinic toxicity if atropine is unavailable or in limited supply. Additionally, Yavuz et al demonstrated reduced myocardial injury and

troponin leak in fenthion-poisoned rats treated with diphenhydramine.[12] In a single-center, randomized, single-blind study by Pajoumand et al found a benefit to magnesium therapy in addition to standard oxime and atropine therapy in reducing hospitalization days and mortality rate in patients with organophosphate poisoning[13]. The mechanisms appear to be inhibition of acetylcholine (ACh) and organophosphate antagonism. Larger randomized studies are needed to demonstrate magnesium efficacy in organophosphate (OP) poisoning.

Possible future interventions include neuroprotective agents used to prevent nerve damage and bioscavengers aimed to prevent AChE inhibition by nerve agents or organophosphate. Investigations into adjunctive and alternative therapies have mostly used animal models and have resulted in variable conclusions.

**Atropine IV/IM (Isopto, Atropair)**

Initiated in patients with OP toxicity who present with muscarinic symptoms.

Competitive inhibitor at autonomic postganglionic cholinergic receptors, including receptors found in GI and pulmonary smooth muscle, exocrine glands, heart, and eye.

**Glycopyrrolate (Robinul)**

Indicated for use as an antimuscarinic agent to reduce salivary, tracheobronchial, and pharyngeal secretions. Does not cross the blood-brain barrier. Can be considered in patients at risk for recurrent symptoms (after initial atropinization) but who are developing central anticholinergic delirium or agitation.

Signs of atropinization might occur earlier with addition of 2-PAM to treatment regimen. 2-PAM administration is not indicated for carbamate exposure since no aging occurs.

Because of risks of respiratory compromise or recurrent symptoms, hospitalizing all symptomatic patients for at least 24 hours in a high acuity setting is recommended. Patients who are asymptomatic 12 hours after organophosphate exposure can be discharged since symptom onset should usually occur within this time frame.

Following occupational exposure, patients should not be allowed to return to work with organophosphates until serum cholinesterase activity returns to 75% of the known baseline level. Also, establishing base line cholinesterase levels for workers with known organophosphate (OP) exposure is recommended[14]Health care providers must avoid contaminating themselves while handling patients poisoned by organophosphates. The potential for cross-contamination is highest in treating patients after massive dermal exposure. Use personal protective equipment, such as neoprene or nitrile gloves and gowns, when decontaminating patients because hydrocarbons can penetrate nonpolar substances such as latex and vinyl.

Use charcoal cartridge masks for respiratory protection when caring for patients with significant contamination.

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