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Organophosphate Toxicity and its Management: A Review

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Abstract

Organophosphorus (OP) compounds are extensively applied for agriculture and domestic pest-control. Self-poisoning of pesticide reports for one-sixth to one-eighth of the world's suicides. OP pesticides inhibit cholinesterase enzymes leading to overstimulation of cholinergic receptors. The analysis of OP poisoning is made on the ground of history of toxicity, odor of pesticides, the distinctive clinical signs and reduced cholinesterase activity. Measurement of plasma cholinesterase is helpful for identification of OP poisoning although it may not directly associate with severity of the poisoning. Atropine remains the major stay of management of OP poisoning with clear proof of benefit if administered efficiently. Pralidoxime can be also applied in suggestive patients but close monitoring is essential. Although several novel adjuvant treatments are tried to attain better result, but their potential benefits are not yet established.

Keywords: Organophosphate toxicity; Epidemiology; Toxicokinetics

Introduction

Organophosphorus pesticide self-poisoning is a foremost clinical and public-health crisis across much of rural Asia of the estimated 5,00,000 deaths from self-harm in the area each year, about 60% are due to pesticide Toxicity [1]. Many Survey approximation that Organophosphorus pesticides are accountable for almost two-thirds of these deaths a total of more than 2,00,000 a year. Deaths from accidental Organophosphorus poisoning are less common than those from intentional poisoning and seem to be more frequent in regions where highly toxic Organophosphorus pesticides (WHO Class I toxicity) are available for e.g. In a large group of Sri Lankan victims intoxicated with WHO Class II Organophosphorus pesticides no deaths outcome from unintentional poisoning [2]. Most victims exposed to organophosphates come into get in touch with insecticides. Organophosphates are used as medications, pesticide, and nerve vectors as a weapon. Symptoms in patients include increased saliva and intensive Lacrimation, nausea, vomiting, diarrhea, contraction of pupils, muscle tremors, sweating and confusion. The onset of sickness is often within minutes, and it can take weeks to disappear [3].

Etiology

Usually exposure of organophosphate pesticides may occur through inhalation, ingestion, or due to crops dermal contact. Crops that farm workers come into contact with also may include organophosphate pesticide such as grapes, lettuce, domestic blueberries, potatoes apple, celery, bell peppers, peaches, strawberries and nectarines.

Epidemiology

Each year it is estimated that 3 million or more people globally are exposed to organophosphates, accounting for about 300,000 deaths. There are around 8000 exposures per year in the United States of America, with very few deaths [4]. While most often the exposure occurs from an agricultural insect killers chemicals, besides household items, such as ant and roach spray, which also contain organophosphate compounds.

Toxicokinetics

Organophosphate molecules can be absorbed in the gastrointestinal tract (ingestion), *via* inhalation or through dermal route. Once absorbed, makes the enzyme inactive, since its molecule binds to an acetylcholinesterase molecule in red blood cells [5]. This leads to an extra amount of acetylcholine within synapses and neuromuscular junctions. At neuromuscular junctions overstimulation of nicotinic receptors can lead to Fasciculations and myoclonic jerks. This finally leads to flaccid paralysis because of the depolarizing block. Nicotinic receptors also are found in the adrenal glands which may cause hypertension, tachycardia, sweating, and a leukocytosis with left shift.

Organophosphate toxicity also produces symptoms based on its function at muscarinic receptors. These effects are usually not faster than the nicotinic receptors because the effects take place via a G-protein-coupled receptor mechanism. Muscarinic receptors are abandon in the sympathetic and parasympathetic nervous system [6]. Because of over stimulation of sweat glands within the sympathetic nervous system causes large amounts of sweating. The parasympathetic effects of organophosphate poisoning can be seen in multiple systems including the heart, exocrine glands, and at smooth muscles. At some point, which is different for each specific compound, the acetylcholinesterase-organophosphate compound undergoes a process called aging. This is a conformational transformation that renders the enzyme resistant to reactivation, making some choices of treatment useless.

History

- Sympathetic and parasympathetic nervous systems both are stimulated by Organophosphates. A distinctive form of clinical situation will engage symptoms of overstimulation of the

parasympathetic system. Though there is an exception is in children, as they typically have a high proportion of symptoms mediated by nicotinic receptors.

- Major symptoms of organophosphate poisonings and the receptor that is responsible for nicotinic signs of acetyl cholinesterase inhibitor toxicity, Mydriasis, Tachycardia, Weakness, Hypertension and Fasciculations.
- The usual mnemonic that captures the muscarinic effects of organophosphate poisonings are Defecation/diaphoresis, Urination, Miosis, Bronchospasm/bronchorrhea, Emesis, Lacrimation, Salivation.
- Hallucinations, headaches, insomnia, Anxiety, confusion, drowsiness, emotional lability, seizures, memory loss and circulatory or respiratory depression are some other symptoms are included in it. The most general cause of death is respiratory collapse stemming from bronchorrhea, broncho-constriction, central respiratory depression or weakness/paralysis of the respiratory muscles. There are other chronic complications though the victim survives the acute poisoning, after contact the Intermediate neurologic symptoms characteristically take place in 24 to 96 hours. Warning signs include neck flexions, weakness, diminished deep tendon reflexes, cranial nerve abnormalities, proximal muscle weakness, and respiratory insufficiency. With supportive care only, these patients can have a absolute return to normal neurologic function within 2 to 3 weeks. An additional later and worst complication is neuropathy. This is connected with very particular compounds that contain chlorpyrifos. Generally this starts as a stocking-glove paresthesia and advances to symmetric polyneuropathy with sagging weakness that starts in the lower extremities and advances to include the upper extremities.

Discussion

Evaluation

When no account of contact or ingestion is known one must have a high clinical doubt for organophosphate poisoning. Identification of acute or chronic organophosphate poisoning is firmly clinical. Garlic or petroleum odors may help in diagnosis of some organophosphates while others have a characteristic smells. If organophosphate poisoning is on the differential but not confirmed a trial of atropine may be employed, if symptoms resolve after atropine, this increases the probability of an acetyl cholinesterase inhibitor poisoning. For identification acetyl cholinesterase activity some labs can directly evaluate red blood cells.

Treatment/management

The very initial step in the treatment of patients with organophosphate poisoning is putting on Personal Protective Equipment (PPE). These patients may still have the compound exposed on them, and you must defend yourself from contact. Secondly, you must decontaminate the patient. This means removing and spoiling all clothing since it may be polluted even after washing. Skin of the patient needs to be flushed with water. To decontaminate the skin dry agents such as flour, sand, or bentonite also can be used. In the case of ingestion, emesis and diarrhea may lessen the quantity of compound absorbed but must never be induced. If the patient presents within 1 hour of ingestion activated charcoal can be administered.

The ultimate management for organophosphate toxicity is atropine, which competes with acetylcholine at the muscarinic receptors. The initial dose for adults is 2 mg to 5 mg IV or 0.05 mg/kg IV for children until reaching the adult dose. If the patient does not counter the treatment, until respiratory secretions have cleared and there is no bronchoconstriction, in this condition double the dose every 3 to 5 minutes. In victims with severe poisoning, it may takes hundreds of milligrams of atropine administered in bolus or continuous infusion over several days before the patient get better. Since atropine only works on muscarinic receptors Pralidoxime (2-PAM) also should be administrated to affect the nicotinic receptors. To avoid worsening of muscarinic-mediated symptoms, treatment of Atropine must be prior to 2-PAM A bolus of at least 20 mg/kg to 50 mg/kg for children or 30 mg/kg in adults should be given over 30 minutes. Fast administration can result cardiac arrest. Following the bolus, a continuous infusion of at least 10 mg/kg/hr to 20 mg/kg/hr for children and 8 mg/kg/hr for adults should be started and may be required for several days.

Conclusion

The diagnosis and treatment of organophosphate toxicity is done with an inter specialized team that consists of an emergency department medical doctor, poison control, anesthesiologist, nurse practitioner, intensivist and other specialists depending on organ system participation. The purpose is to prevent further absorption via the skin, eyes, or lungs. The management should follow the trauma protocol with the first priority on the airways. Symptomatic patients need to be observed in the ICU. Both atropine and Pralidoxime can be applied in suggestive patients but close monitoring is essential.

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