Organophosphate Intoxication

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Abstract

Organophosphate compounds are potent inhibitors of cholinesterase; most of these compounds are used as insecticides, pesticides, or drugs. Like many other countries, poisoning with organophosphate compounds is one of the most important causes of intoxication in Taiwan. Manifestations depend on the agent and its concentration. The mode of contact in organophosphate poisoning varies, as these compounds are absorbed efficiently by oral, dermal, conjunctival, gastrointestinal, and respiratory routes. Organophosphates strongly inhibit both true acetylcholinesterase and pseudocholinesterase which leads to accumulation of acetylcholine at the synapses. As a result, muscarinic and nicotinic symptoms appear. The onset of the signs and symptoms of organophosphate poisoning varies with the route and degree of exposure. Treatment of poisoning with organophosphate is directed toward four goals: (1) decontamination, (2) supportive care, (3) reversal of acethylcholine excess at muscarinic sites, and (4) reversal of toxin binding at active sites on the cholinesterase molecule. Due to potential prolonged effects of acetylcholinesterase inhibition, most individuals with significant exposures require hospital admission and regular follow up. *(Ann Disaster Med. 2005; 4 Suppl 1:S23-S28)*

Key words: Organophosphate; Antidote; Toxicology; Cholinesterase

Introduction

Organophosphates (OP) are used widely in agriculture, horticulture, and veterinary medicine. These insecticides also are used domestically and in public hygiene to control vectors of disease. Some OP compounds (e.g., malathion) are used to treat human infestation with scabies, head lice, and crab lice. Examples of OPs include insecticides (malathion, parathion, diazinon, fenthion, chlorpyrifos), nerve gases (soman, sarin, tabun, VX), ophthalmic agents (echothiophate, isoflurophate), and antihelmintics (trichlorfon).[1] The primary action of OP insecticides on insects, and the source of their potential toxicity to humans, is a consequence of their ability to inhibit the enzyme acetylcholinesterase (AChE).[2,3] The result is an acetylcholine (ACh) excess syndrome.

Pathophysiology of Toxic Effects

OP can affect almost all organ systems but their high lethality stems from their capacity to cause paralysis of the respiratory muscles and severe depression of the central nervous system

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Received: Aug 25 2005. Revised: Aug 30 2005. Accepted: Sep 5 2005.
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