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FEATURE ARTICLE

PEER REVIEWED

Recognizing Dangerous Poisonings in Primary Care: Part 5, Pesticides

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The 5 most common causes of poisoning-related fatalities in the United States are antidepressants, antihistamines, cardiovascular drugs, opioids, and pesticides.[1] Drug poisonings, particularly mild cases, are often exceptionally difficult to recognize. Slightly dilated pupils, mild tachycardia and hypertension, slight fever, and tachypnea are all constitutional symptoms that fit myriad working diagnoses, but autonomic instability is a hallmark of drug poisonings. Motor stiffness and hyperreflexia are frequently seen with a number of drug poisoning syndromes, although not with pesticide poisoning.[2,3]

This 6-part review article series helps sort out some of the more common symptoms, interactions, and therapeutic considerations in the clinical approach to a patient whom you suspect may be experiencing the effects of the most common types of poisoning.

This article, the fifth in the series, covers pesticide poisonings. Other articles in the series cover antidepressants, antihistamines, cardiovascular drugs, and opioids, and one article specifically covers serotonin syndrome.

The American Association of Poison Control Centers' National Poison Data System annual report for 2011 reported 89,000 pesticide exposures, representing 3.26% of all reported exposures for that year. This marked a slight decrease in pesticide exposures from the previous year, but pesticide exposure retained its ranking as the 10th most common reported exposure. Internationally, pesticide poisoning is one of the most common causes of fatal overdoses due to the easy accessibility of these agents.[4]

Pesticides are a large group of diverse chemicals and are too numerous to discuss in detail in this review article. This article will focus on the 2 most common pesticide classes in current use: organophosphates (OPs) and organochlorines (OCs). Both classes have a distinct mechanism of action, and both can be fatal in high enough concentrations.[5]

Organophosphate Poisoning

OPs are a diverse group of chemicals that are used for a wide variety of commercial and industrial applications, including insecticides, herbicides, and chemical weapons (nerve gases). OP exposure can be secondary to cutaneous absorption, ingestion, or inhalation.[6]

The primary mechanism of action of OPs is the inhibition of acetylcholinesterase. This results in an accumulation of acetylcholine in the central and peripheral nervous systems, causing an overstimulation of the muscarinic and nicotinic receptors.[7] The muscarinic effects of acetylcholine can be remembered using the classic mnemonic DUMBBELLS (diaphoresis and diarrhea; urination; miosis; bradycardia, bronchospasm and bronchorrhea; emesis; excess lacrimation; and salivation).[8] The nicotinic effects can cause hypertension, tachycardia, generalized weakness, and muscle cramps.[5]

The treatment of patients with a suspected exposure to an OP should begin with decontamination of the patient and protection of the staff. The staff should immediately put on masks and neoprene gloves. The patient should be immediately disrobed and rinsed with a soap and water solution.[6]

The primary treatment of OP exposure is atropine and pralidoxime (2-pyridine aldoxime methyl chloride, or 2-PAM). Supportive care is the mainstay of treatment initially.[9] In severe poisonings, airway protection may be necessary to prevent aspiration from the increased secretions and to provide positive airway pressure. The standard doses of atropine (0.2-0.4 mg) are usually insufficient in OP exposures, and doses as high as 6 mg (or as a continuous infusion) may be required to alleviate the clinical symptoms of the exposure.[5] Tachycardia as a result of atropine use is not a reason to limit or stop subsequent doses, since the main concern in OP poisoning is respiratory failure from excessive airway secretions. Pralidoxime is used as an antidote to reverse the muscle paralysis resulting from OPs. It is a nucleophilic agent that reactivates phosphorylated acetylcholine by binding to the OP molecule and displacing acetylcholine, but it is not effective once the OP compound has irreversibly bound to the acetylcholine. Current recommendations are to administer pralidoxime within 48 hours of exposure. These patients all require admission until their symptoms resolve.

Organochlorine Poisoning

OCs are widely used around the world despite that a large number of these chemicals have been banned.[10] These compounds tend to be highly toxic and are composed of carbon, hydrogen, and chlorine. They tend to be insoluble in water and to accumulate in water supplies.

OCs are stored in lipids and accumulate in the liver and brain and do not break down easily. This can lead to long-term accumulation of these compounds that can be passed up the food chain to humans.[11] Some of the more commonly known compounds are dichlorodiphenyltrichloroethane (DDT), lindane, and toxaphene. OC exposure occurs through oral ingestion or inhalation but not via the skin, since these compounds have very poor cutaneous absorption.[12] Patients can present with a variety of symptoms, including central nervous system effects such as agitation, lethargy, hallucinations, and seizures.[13] These compounds are also known to have pulmonary effects and can cause significant dyspnea and cough. Long-term exposure can lead to pulmonary fibrosis, hepatotoxicity, and renal toxicity.[14]

Treatment for suspected OC exposure should begin with the immediate decontamination of the patient. Health care workers need to be cautious and observe universal precautions during the care of these patients. A careful history should be obtained from the patient or from any witnesses if the patient is unable to present a history.[15] If the ingestion has occurred less than 1 hour prior to presentation, then gastric decontamination can be attempted using activated charcoal.[16] The mainstay of care is supportive. Currently, no known antidotes exist for OC

charcoal. The mainstay of care is supportive. Currently, no known antidotes exist for OC exposure. Clinical symptoms should be treated, benzodiazepines can be administered for agitation, and 0.9% sodium chloride boluses as well as intravenous phenylephrine should be utilized for pressure support. B-blockers, specifically propranolol and esmolol, can be utilized to treat ventricular dysrhythmias. Seizures can be treated with either benzodiazepines or with phenytoin or fosphenytoin.[11,17] If hepatotoxicity is suspected, *N*-acetylcysteine (NAC) should be administered. Currently, there is no evidence that demonstrates improved outcomes with NAC; however, there also is no evidence of worsened outcomes with NAC.[18-20] Patients with mild exposures should be observed for 6 to 8 hours; if no clinical symptoms are present, then the patient can be safely discharged home. All other exposure cases should be admitted for inpatient treatment.

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