

What Physicians Don't Know About Occupational Exposure to Pesticides

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"Why hasn't my physician told me more about the pesticide hazards I am facing?"

"Why is s/he reluctant to diagnose my illness as pesticide-related when it seems at least an obvious possibility?"

These are questions frequently asked by workers when they first learn about the possible hazards they face from exposure to pesticides on their job. One answer is that their physician may not have been trained to investigate occupational exposure as a cause of illness.

In a 1979 survey of U.S. medical schools, 70% of the responding schools indicated they required no formal instruction in occupational or environmental medicine. Among the 30% that did require such instruction, the median time required was four hours during the four years of medical education. In a repeat survey conducted in 1984, 54% of the schools included such instruction, but the median time required was still four hours (Levy 1985).

Acute, Severe Poisoning

What most physicians know about pesticide toxicity is limited to the specific signs of acute, severe poisonings. Some chemicals produce characteristic physiologic changes which make diagnosis easier, but almost all of these signs and symptoms occur only after a substantial exposure. In cases of direct exposure to large amounts of a pesticide (e.g. following a spill, accidental ingestion, or direct spray by a helicopter), some effects will be so specific that they suggest the diagnosis of pesticide poisoning.

Not very many categories of pesticides have specific "signs," however, and even these signals may be misinterpreted if the link to pesticide exposure is not made. For example, a sign of moderately severe organophosphate or carbamate poisoning is miosis, or pin-point pupils. If pesticide exposure is not mentioned by the patient or by the person who brings the patient to the emergency room or clinic, most physicians would initially suspect narcotics abuse because it is the most common reason for this finding in a patient. When pesticide exposure is mentioned, and the symptoms are severe, many nurses and physicians will recognize the presenting symptoms as signs of pesticide poisoning or will make use of a poison control center and other resources to investigate the possibility of pesticide poisoning.

Chronic, Low-level Poisoning

Acute severe poisonings are relatively rare, however, in comparison with low-level pesticide exposures at work or in home and garden use. The effects of low-level exposure are much more difficult to diagnose, for a number of reasons:

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1) The symptoms are almost always non-specific, meaning that they could be caused by many different chemicals, by an influenza or cold, by physical exhaustion or even psychological stress. Mild organophosphate exposure may only produce headache, fatigue, weakness, nausea and sweating, all those being classic signs of a beginning bout of flu. Dermatitis caused by a pesticide could easily be diagnosed as a reaction to a soap or plant at home rather than a pesticide in the workplace. Medical students learn to "look for the zebras" (i.e. think of exotic explanations for common findings) while they are in school, but everyday practice in the real world teaches most physicians that "if it looks like a horse, it probably is not a zebra." In other words, if there is a logical, common explanation for a set of symptoms, why try to dig up another explanation?

2) In most cases it will not be easy for the physician to "prove" the diagnosis. Making a diagnosis means both a) demonstrating the probability that a certain agent (e.g. a pesticide) caused the illness, and b) ruling out other causes (e.g. demonstrating that the patient doesn't have the flu or a cold). Since many illnesses like a cold or flu can't be conclusively ruled out in most situations, diagnosis depends upon making a strong case for the probability that pesticides caused the illness.

Biological testing for pesticides in the blood or urine is relatively difficult and very expensive (the usual method is gas chromatography and it may cost several hundred dollars per test), and not very helpful in low-level exposures. Exposure to organophosphate or carbamate compounds is measured by the activity of the enzyme cholinesterase in blood. The interpretation of this test is difficult in mild or moderate exposures, however, because there is a wide range of variation in enzyme activity between individuals. A mild effect in one person is almost impossible to detect without a baseline measurement for that person prior to the exposure for comparison. As a result, a physician who says "I think this patient has a headache and nausea because she worked on a railcar that was shipping pesticides" has no way of demonstrating why that patient didn't just have a mild case of flu.

3) In some cases the exposure occurred a long time before the onset of symptoms, or the symptoms have existed for a while before the patient realizes that a past exposure might have caused them. Again, unless there is some symptom or finding which clearly demonstrates the link with the pesticide exposure in the past (for example, a peripheral neuropathy developing several weeks after exposure to certain organophosphates), this is difficult to diagnose. Even the most sympathetic physician, one very interested in pursuing occupational etiologies (causes), has a tough time defending a diagnosis in cases like this.

4) Very little research has been done on the clinical toxicology of pesticides, and even less is published in medical journals and texts. When a physician is puzzled by a clinical situation, she or he turns first to textbooks in the office. If a case is very unusual