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Dear Steve:

I am sending the enclosed report in response to the December 10, 1993 notice in the MMWR regarding the workers' family protection act. Our illness registry contains several additional case reports that might be worthwhile summarizing in a brief MMWR article, although the most recent episode dates from 1990 1991 and the oldest from 1982.

Sincerely,

Michael O'Malley

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Childhood Poisoning Associated with the Insecticide Aldicarb

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INTRODUCTION

Physical Properties and Uses

Aldicarb (Temik®) is a white crystalline solid with a water solubility at room temperature of 0.6%, a vapor pressure of $1x10^{-4}$ mm Hg, and a sulfur-like odor.[1] It was first introduced in the 1960's for systemic control of a variety of insects, mites, and nematodes.[2] The principal uses in California have included cotton (79% of all pounds of aldicarb used), sugarbeets, ornamental flowers, and potatoes.[3]

Toxicity

Structurally aldicarb is an n-methyl carbamate cholinesterase inhibiting insecticide with an extremely high acute toxicity (oral LD50 ranging from 0.46 to 1.23 mg/kg in the rat; and dermal LD50 from 3.2 to >10 mg/kg in the rat and 5-20 mg/kg in the rabbit).[4] Signs of carbamate poisoning usually occur 15-30 minutes after exposure and may include lacrimation, urination, abdominal pain and cramping, defecation, muscular fasciculations and tremors, constriction of pupils, vomiting, ataxia, and bradycardia. In severe cases, these signs are often followed by muscular weakness and spasms that often progress to convulsions.[5] Signs of recovery occur within one to several hours after exposure to the pesticide[6] due to the the rapid breakdown of the carbamate-cholinesterase enzyme complex. *In vitro* breakdown of the enzyme-inhibitor complex may also occur, making the detection of cholinesterase inhibiton problematic in many cases of symptomatic illness.[7]

Case Report

California law requires physicians to report pesticide related illnesses to the Pesticide Illness Surveillance Program (PISP) by notifying the local county health department within 24 hours of recognizing a suspected case. From 1982 through 1990, the PISP reported 47 cases of aldicarb related systemic illnesses, with the majority (26) of the cases resulting directly from aldicarb applications. Non-occupational exposures, mostly involving aldicarb contaminated produce, also accounted for a significant number of cases (17). The latter group included only two cases from the 1985 aldicarb watermelon poisonings, estimated to involve more than a thousand probable cases in California, Washington, Oregon, and Canada.[8,9,10]

This report describes a case childhood poisoning associated with the carbamate pesticide aldicarb that occurred in 1990.

CASE REPORT

On April 1, 1990, M.L., a three year old Hispanic girl, was playing in her yard with her brother and cousins. Her father worked for a Tulare County dairy farm and the family lived on the dairy property in a mobile home adjacent to a block of land leased by the dairy to a local cotton grower. A tractor and trailer loaded with pesticide and cotton seed was parked about fifty feet from the door of the home.

At approximately six in the evening, M.L. complained to her companions of a stomach ache and then lost control of the right side of her body. The boys took her to the back door of their home and yelled for their parents. The child was then foaming at the mouth and had lost most control of her body. The mother subsequently reported that she suspected the child had ingested some pesticide or cotton seed from the back of the trailer. Her parents drove her to the a rural fire station 5-8 miles away. While waiting for the ambulance to arrive, she was noted by her mother to have lost control of her bowel movements. The ambulance arrived at 6:36 p.m. and she was taken to the local district hospital.

Upon arrival at the emergency room at 7:00 p.m., the child was lethargic and had a very weak response to painful stimuli. Her pupils were pinpoint and not reactive to light. Her breathing was labored, there was copious thick white sputum in the respiratory tract, and auscultation of the chest revealed bilateral moist rales. The examining physician noted fasciculations on her left hand and around her eyes. Following a doses of 0.55

and 0.75 mg of intravenous atropine there was a marked decrease in airway secretions and clearing of her breath sounds. An additional 0.7 mg of atropine was given at 7:50 p.m. At 7:55 p.m. a 750 mg intravenous piggyback of protopam hydrochloride was administered and blood sent for cholinesterase enzyme levels.

After approximately an hour of observation, the child became increasingly lethargic. She had intermittent episodes apnea for a period of 30 minutes and increased fasciculations around her mouth and arms lasting approximately fifteen minutes. She received an 0.75 mg of atropine at 8:20 p.m. and additional dose of 0.5 mg at 8:53. Her breathing became more requlars and she more responsive to her mother. At 9:31 her plasma cholinesterase levels were reported as 3.8 μM/ML, with a normal population range > 2 μM/ML; RBC cholinesterase was 9.3 with a normal value of >8 μM/ML. No followup samples were taken and no baseline levels were available for comparison.

At 9:21 p.m. the child was transferred to another district hospital under the care of her family pediatrician. At the time of admission she was alert, but somewhat subdued. Her physical examination was unremarkable except for the presence of slight expiratory wheezing. Within 24 hours of hospitalization, the child was alert and requesting liquids. She was discharged on April 3 in stable condition.

Vomitus samples taken on April 2, analyzed by the pesticide manufacturer,
showed 0.03 ppm of aldicarb. A sample of nasogastric aspirate collected on April 3 and analyzed at the CDPR chemistry lab (with a minimum detection limit of 0.5 ppm) showed no detectable level of aldicarb. The CDPR chemistry lab also received a urine sample collected on April 2 with a request to analyze for aldicarb metabolites that was negative for aldicarb (with a minimum detection limit of 0.1 ppm).

On April 2, 1990 Tulare County Agricultural Commissioner's Office began investigating the incident. It was determined that the tractor parked near the child's house had a box of cotton seed and a box of Temik® 15-G granules (aldicarb). A sample from the latter box confirmed that it contained 14.5% concentration. Samples of soil taken 15 feet from the house showed 1.84% aldicarb.

COMMENTS

The sudden onset in a previously healthy child of excessive salivation, diarrhea, bronchoconstriction, miosis, altered sensorium, muscle fasciculations, and muscular incoordination appropriately suggested to the treating physician that the child had been poisoned with a cholinesterase inhibiting insecticide. This conclusion was strengthened by the positive clinical response to the administration of intravenous atropine. She suffered a brief period of near apnea within an hour of receiving an intravenous dose of 2-PAM HCL, but restabilized following repeated doses of atropine. Over the following 24 hours she recovered nearly completely, suggesting that the cholinesterase inhibitor was a carbamate rather than an organophosphate insecticide. The finding of RBC and plasma cholinesterase activitity within the laboratory normal range at the time of her initial clinical illness is also more consistent with the diagnosis of carbamate than organophosphate poisoning because of the inherently unstable nature of the carbamate-cholinesterase complex.[11,12,13] Carbamate exposure is suggested by the possible adverse clinical response to the administration of protopam hydrochloride.

Investigation subsequently demonstrated that the child's yard was contaminated with the carbamate insecticide aldicarb. The finding of 0.03 ppm aldicarb in the nasogastric aspirate on the second day in the hospital confirms that at least some of the insecticide was ingested, but leaves in question whether ingestion was the principal route of exposure. Like other carbamates, aldicarb is readily absorbed from both the gastrointestinal tract and the skin.[14] In this instance, both routes of exposure may have occurred.

Children who live or work in agricultural settings deserve special consideration in evaluating the risks associated with the farm environment. In addition to the well described exposures to the hazards of farm machinery,[15,16] children living on farms have many opportunities for exposure to highly toxic farm chemicals. Because of the seasonal nature of agricultural work, serious poisonings may result from the presence of unexpected hazards in the child's familiar environment.

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