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Acephate: A Safer Pesticide Alternative?

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Background:

Acephate is a weak organophosphate (OP) that is hydrolyzed in insects to methamidophos (MP), a potent OP. Rats metabolize acephate primarily to des-O-methylacephate as MP is thought to inhibit its own formation in mammals. This is the basis for the presumed selectivity for insects. The effects of AP in humans are poorly characterized and the role of treatment with antimuscarinic and oxime therapy is unknown. We present a case series of three patients with acute ingestion of acephate.

Case Reports:

Patient 1 is a 32-year-old female who presented with erratic behavior, diaphoresis, vomiting, and rapid heart rate after ingesting acephate mixed in water following an altercation. She had "hypoactive delirium", confusion, diarrhea, sialorrhea, tachypnea (33 bpm), hypothermia (94.50°F), tachycardia (117 bpm). Lung exam was normal. The patient was started on pralidoxime. She developed respiratory secretions requiring atropine, acidosis treated with bicarbonate infusion and hypotension refractory to IV hydration requiring norepinephrine infusion. Resolution of cholinergic symptoms was noted 96 hours from ingestion.

Patient 2 is a 16-year-old female who presents after ingesting "a spoonful" of acephate with suicide intent. She has pallor, diaphoresis, pinpoint pupils, tachycardia, emesis, diarrhea, abdominal pain, and confusion. The patient later had generalized weakness, flat affect, and poor muscle tone. She was treated with pralidoxime and medically cleared at 70 hours post ingestion. Pseudocholinesterase was 2.5 (Ref: 4.9-11.9)

Patient 3 is 50-year-old female who presents with vomiting, dilated pupils, tachycardia, lethargy, and acidosis after reported ingestion of boric acid and acephate mixed with water. The patient did not develop bronchorrhea however she received atropine for nausea 52 minutes after ED arrival. She was treated with pralidoxime. Symptoms resolved by 23 hours post ingestion.

All three patients became symptomatic less than 5 hours after reported ingestion.

Case Discussion:

Acephate exposures are poorly characterized and previously thought to be unlikely to produce significant toxicity due to differing metabolism between insects and mammals. The three patients with varied presentations all exhibited cholinergic symptoms. However, some of patient 3's symptoms could be attributed to boric acid ingestion.

Conclusion:

Acephate can cause significant and varied presentations of cholinergic toxicity in humans.