ILLUSTRATIVE CASE

Pediatric Pesticide Poisoning: A Clinical Challenge
Stefani Samples, MD,* Alexander Eason,* Henry Wiles, MD*

CASE

The patient in this case was an otherwise healthy 12-year-old boy who presented to our hospital for monitoring after exposure to a pesticide. His medical history was significant only for attention-deficit/hyperactivity disorder, for which he took daily stimulant therapy. In the trailer he shared with his grandmother, an unknown amount of the pesticide tablets were accidentally spilled indoors. In an attempt to clean it up, they vacuumed up the substance, which reportedly caused the vacuum to explode, dispersing the pesticide in the air. When both the patient and his grandmother began vomiting a few hours later, they were taken to a local emergency department by family members. The grandmother experienced cardiac arrest and expired in the local emergency department within 12 hours of exposure. The patient was then transferred to our tertiary medical center for additional monitoring because of his pesticide exposure. He was initially monitored with no concerning symptoms. His heart rate was mildly elevated at rest, averaging 110 to 130 beats per minute, but his blood pressure was normal for age and height. Less than 48 hours after initial pesticide exposure, he developed a new, prominent gallop rhythm on physical examination. An echocardiogram was performed, which demonstrated a mildly dilated left ventricle with severely diminished left ventricular systolic function. The fractional shortening was 22%, and the ejection fraction was 36%. The electrocardiogram (ECG) was also abnormal, demonstrating sinus tachycardia, left axis deviation, poor R-wave progression, and diffuse T-wave flattening (Fig 1). All intervals, including QRS duration (88 msec) and QTc (444 msec), were within normal limits.

Question
What are the findings in dilated cardiomyopathy?

Discussion
As a whole, cardiomyopathy is a diverse class of cardiac diseases. Dilated cardiomyopathy is characterized by the development of dilated, poorly functional ventricles with normal wall thickness not precipitated by an abnormal cardiac preload or afterload.1,2 Dilated cardiomyopathy can be accompanied by cardiac arrhythmias and symptoms of heart failure. The ECGs of patients with dilated cardiomyopathy may be normal but may also demonstrate varying types of heart block, atrial fibrillation, or even ventricular arrhythmias.1,2 Echocardiography demonstrates increased ventricular dimensions, ventricular hypokinesia, and a decreased fractional shortening. Symptomatic relief can often be achieved through the use of diuretics to decrease the volume load placed on the heart. Angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, aldosterone antagonists, vasodilators, and β-blockers are all associated with increased survival and are used in almost all patients with dilated cardiomyopathy, from those with severe heart failure to those with asymptomatic left ventricular systolic dysfunction.2

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After an extensive literature review and discussion with several national toxicologists, we determined that this was a documented effect of exposure to aluminum phosphide pesticides. This was also supported by review of the patient’s admission laboratory values, which demonstrated a metabolic acidosis (bicarbonate 15 mEq/L) and mild hyperglycemia (glucose 147 mg/dL), both of which quickly resolved on subsequent testing. To ensure this was not a coincidental finding of cardiomyopathy of another cause, a cardiomyopathy evaluation was undertaken. Acute viral titers, including herpes simplex virus, Epstein-Barr virus, echovirus, cytomegalovirus, coxsackievirus, and adenovirus, as well as convalescent titers obtained 20 days later, were negative with the exception of a positive mumps immunoglobulin G, thus indicating an appropriate immune response to vaccines without any additional evidence of acute infection. The patient’s erythrocyte sedimentation rate at diagnosis was normal (8 mm/hr). C-reactive protein was only mildly elevated (1.063 mg/dL). Thyroid studies were normal, as were profiles of amino acids, acylcarnitines, and carnitines. An initial troponin was elevated (1.308 ng/mL), but this quickly normalized.

Supportive treatment was then initiated. Several national toxicologists recommended placing the patient on intravenous (IV) fluids, which could aid in pesticide excretion. Although asymptomatic, he was also started on afterload reduction using lisinopril due to his diminished left ventricular systolic function. A 24-hour Holter monitor returned with normal findings, and a follow-up echocardiogram performed 3 days later demonstrated normal left ventricular systolic function with an improvement of his fractional shortening (32%) and ejection fraction (61.3%). Another echocardiogram done during his outpatient follow-up 20 days later continued to demonstrate normal left ventricular systolic function.

**Question** How frequently does aluminum phosphide exposure occur, and what are its biological effects?

**Discussion**

Aluminum phosphide poisoning is a rarely reported entity in the United States. Most reports are due to job-related exposure or suicidal attempts occurring outside the United States. Childhood exposure is even less common, with the exception of older teenagers who ingest aluminum phosphide containing pesticide pellets with suicidal intent. The national poison control centers data from 2013 reported that 3.27% of reported exposures were due to pesticides in general, and only 0.006% were due to aluminum phosphide exposure, the majority of which occurred in adults. There have been rare case reports of unintentional aluminum phosphide poisoning of entire families that have reported fatalities due to acute cardiopulmonary collapse in children aged 15 months up to 6 years. Both of these reports demonstrated that the cardiac deterioration could occur far removed from the initial exposure. These reported families had repeated exposure to the aluminum phosphide-containing pesticides before the poisoning symptoms were recognized, which differs from our case.
Aluminum phosphide is an inorganic compound commercially available as pesticides that are used to protect crops from insects and rodents. Commercial names for aluminum phosphide include Fumtoxin, L-Fume, Tri-Tox, Fumiphos, Phostoxin, Fumex, Gastoxin, and Quik-Fume. It is manufactured in dust, granular, and pellet forms. These readily react to form phosphine gas (PH₃) after coming into contact with water, moisture, or the acidic content in the human stomach. Once inhaled or ingested, phosphine gas leads to the production and accumulation of reactive oxygen species (ROS) at the cellular level and subsequent lipid peroxidation. It is this extensive ROS generation that is believed to be responsible for the wide systemic toxicities of phosphine gas exposure. Specifically, these effects cause alterations in the cardiac transmembrane action potentials, which lead to dysrhythmias. They may also cause ischemic effects that can induce focal areas of necrosis as well as cardiac failure.

Phosphide exposure is known to rapidly affect multiple organ systems including the respiratory, cardiovascular, hepatic, renal, hematologic, and gastrointestinal systems. Early symptoms of acute exposure can vary on the exposure route and dose, but typically include fatigue, dyspnea, nausea, vomiting, abdominal pain, hypotension, and tachycardia. Progression of illness can be rapid and may lead to organ failure of any of the affected organs systems, but the most common effects include acute respiratory failure and cardiac shock. Other potential systemic effects of exposure include acute hepatitis, acute renal failure, and disseminated intravascular coagulation. Multiple metabolic derangements have also been described including abnormalities in sodium, potassium, and/or magnesium levels, lactic acidosis, and hypoglycemia.

**Question** What are the available treatments for aluminum phosphide exposure?

**Discussion** Perhaps because of the wide range of cytotoxic effects that phosphide exposure has, no antidote has yet been identified. If exposure occurs, the Centers for Disease Control recommends decontamination as soon as possible by experienced personnel. Treatment remains largely supportive, and several therapeutic strategies have been proposed to aid in the management of acute exposure. Maintaining renal perfusion with IV fluids may aid in phosphine elimination. Digoxin and other inotropic agents have had some effect in treating cardiovascular collapse and ventricular dysfunction in these cases. In 1 case report, calcium gluconate and magnesium sulfate were used for cardiac membrane stabilization. In a study of children after ingestion of aluminum phosphide pesticides, magnesium sulfate was associated with better survival. Other proposed treatments include high doses of vitamin C as an antioxidant or IV magnesium and/or N-acetylcysteine to replenish intracellular glutathione stores and reduce systemic ROS levels; however, none of these methods have as yet demonstrated themselves to be particularly effective. There is a report of using of an intra-aortic balloon pump successfully for a patient with myocarditis and cardiac shock whose clinical condition initially worsened despite inotropic support. Our patient received only minimal supportive treatment with IV fluids and lisinopril therapy before recovery. Several patients with phosphide-induced cardiomyopathy have reportedly experienced improvements of their left ventricular function over the course of their hospital stay, as our patient did. It is likely that this rapid recovery is due to clearance of phosphide from the patient’s system.

**CONCLUSIONS** Although aluminum phosphide exposure is rare in the United States, it presents a treatment dilemma given the paucity of definitive information available in the literature, especially in relation to children. Although there are no current consensus statements, supportive treatment appears to be the best choice for now. However, the best treatment is always the prevention of exposure by limiting usage of these pesticides, especially in the presence of children, and appropriately storing and disposing of them. If a pesticide spill occurs in the home or workplace, families should contact the national poison control hotline (1-800-222-1222) for instructions on appropriate cleanup and treatment as needed.

**REFERENCES**


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