INTRODUCTION

Aluminum phosphide, a very effective rodenticide and insecticide is commonly used to protect grains and tobacco stores during storage and shipment.1,2 It is formulated either as a pellet or a tablet.1 Upon exposure to water, it releases phosphine gas which is the active agent and has toxic potential. The chemical reaction responsible for this is as follows:

$$\text{ALP} + 3\text{H}_2\text{O} \rightarrow \text{Al(OH)}_3 + \text{PH}_3$$

Phosphine gas (PH3) is responsible for these toxic effects of Aluminum Phosphide (ALP).1,3 Phosphine is a colorless gas and typically odorless when pure. However, when it is mixed with other substances it has a fish or garlic odor. Phosphine gas is highly toxic to insects, burrowing pests, humans, and animals.1 Clinical manifestations of phosphine toxicity depend upon the amount of gas to which the person is exposed. This is expressed as parts per million (ppm).4 Cardiac toxicity is the most common cause of mortality in most of cases. There is no specific treatment of phosphine poisoning, and fatal outcome is common.5,6 Occupational exposure can occur in industry workers. Aluminum phosphide (ALP) has been reported as a cause of suicidal exposure in adults and also accidental poisoning in children in India.5 But there is no available data from Pakistan.

We report accidental inhalational exposure of 2 families after house fumigation with a fatal outcome.

CASE REPORT

Accidental Phosphine Gas Poisoning with Fatal Myocardial Dysfunction in Two Families

Saleem Akhtar1, Arshalooz Rehman1, Surraya Bano2 and Anwarul Haque1

ABSTRACT

Aluminum phosphide is commonly used as a rodenticide and insecticide and is one of the most fatal poisons. The active ingredient is Phosphine gas which inhibits cytochrome oxidase and cellular oxygen utilization. The clinical symptoms are due to multorgan involvement including cardiac toxicity which is the most common cause of mortality. Severity of clinical manifestations depends upon the amount of the gas to which a person is exposed. There is no specific antidote available. High index of suspicion and early aggressive treatment is the key to success. We report 2 cases of aluminum phosphide toxicity in 2 families due to incidental exposure after fumigation.

Key Words: Aluminum phosphide. Cardiac dysfunction. Fumigation.

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be Aluminum Phosphide (ALP). The third child was fortunate and had mild cardiac dysfunction which recovered completely after 3 - 4 months of supportive cardiac treatment.

**Case 2:** A toddler aged 3 years and an infant aged 379 1980; Journal of the College of Physicians and Surgeons Pakistan 2015, Vol. 25 (5): 378-379 were all right few hours back. Family gave the history of fumigation one day prior to onset of symptoms. The protocol for fumigation was the same as described in the above case. The older child was sick and despite adequate fluid resuscitation continued to deteriorate. He was tachycardiac, tachypnoeic and developed metabolic acidosis despite fluid resuscitation. He was treated as sepsis and antibiotic coverage was added. He, however, continued to deteriorate. So his electrocardiogram (ECG) and echocardiogram and cardiac enzyme (Troponin I) were investigated. ECG showed sinus tachycardia and ST-T segment changed indicating cardiac injury. Echocardiogram showed severe cardiac dysfunction. His Troponin I was also increased. He was started on intravenous inotropes (dopamine and milrinone) and shifted to PICU. Within half an hour of being shifted to intensive care unit the patient went into ventricular fibrillations and expired despite attempted resuscitation. The other child was aggressively managed in the ER with inotropic support and was electively intubated in the ER. His work up also showed moderate myocardial dysfunction on echocardiogram, sinus tachycardia on ECG along with ST-T changes and elevated cardiac enzymes. With aggressive therapy he was stabilized and was discharged in a week time from intensive care unit. His myocardial function fully recovered in 3 months time and at 1-year follow-up he is doing fine and is off supportive therapy.

**DISCUSSION**

The death of these children was due to myocardial dysfunction secondary to phosphine inhalation as shown by decreased ventricular function, electrocardiographic abnormalities, and elevation in blood concentrations of Troponin and creatine kinase isoenzyme MB. Clinical findings reported by various authors in acute phosphine poisoning include headache, vomiting, cough, depressed mental status, pulmonary edema, hypotension, cardiac dysrhythmias, liver and kidney failure, and severe gastritis. The children in these cases also presented with nausea and vomiting and later developed the clinical features of cardiovascular toxicity. The cardiovascular system toxicity of phosphine is quite varied and extensive. It ranges from mild hypotension to complete cardiovascular collapse and is the cause of mortality as is seen in these cases. The exposed children in these cases also developed manifestations of cardiovascular collapse and could not be saved despite maximum available supportive treatment. Unintentional poisoning from inhalation of phosphine gas is rare. However, occasional occupational exposure and suicidal ingestions are reported. Occupational exposure limits for phosphine gas have been established. The National Institute for Occupational Safety and Health occupational exposure has established limits for phosphine gas at 0.3 ppm while levels equal to or more than 50 ppm are described as "immediately dangerous to life or health." Mortality rate as high as 77% has been reported. Management of the patients affected with phosphine is mainly supportive as was done in these patients. There is no accepted specific treatment. A variety of manoeuvres have been suggested as possibly improving outcome; including administration of magnesium, N-acetylcysteine, pralidoxime, or trimetazidine, as well as a combination of insulin and glucose administration along with hyperventilation. Few case reports of use of vegetable oil and coconut oil have also been reported. However, at present evidence is insufficient to advocate use of any of these therapies as a component of care. Organophosphates are generally considered the most likely agent when history of fumigation is present. However, other agents should be considered especially when there is evidence of serious cardiovascular compromise along with history of fumigation.

**REFERENCES**