Case Report

Phosphine inhalation, case series of domestic unintentional poisoning commonly used as anti-bed bug chemical from a low income country

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ABSTRACT

Phosphine poisoning is an uncommon but hazardous public health issue. It is widely used chemical compound as insecticides domestically and exposure to this chemical is mostly accidental especially in children, which may lead to fatal symptoms if not managed immediately. Previous studies have explored the mechanism of its deleterious effect. However, literature on presentation of phosphine poisoning is sparse. Medical records, from January 2005 to September 2013, with patients presenting to ED of Aga Khan University Hospital were reviewed after successfully taking ERC approval from the hospital. Patients with confirm phosphine poisoning were identified and further details were noted from their medical records. We identified seven cases of phosphine poisoning during the study period. Majority patients presented with generalized weakness, diarrhea, vomiting and drowsiness after exposure to phosphine containing insecticides, mostly exposed within past two days of symptoms and presentation. Six out of seven patients were successfully managed and discharged while one of the patient couldn't be saved. The objective of this study was to investigate various presentations of phosphine poisoning to Emergency Department (ED) in a low-income setting in Karachi, Pakistan and bring it to attention of the need to replace insecticides with a safer chemical compound and keep children out of reach from such toxins. Moreover increase awareness program and proper labeling and precautions should be written on all commonly sold pesticides and the constituent of hazardous compounds they contain.

Keywords: Phosphine, Insecticides, Accidental poisoning

INTRODUCTION

Acute poisoning is a public health issue worldwide especially in low income countries. Almost 300,000 people die due to pesticide poisoning every year globally.1 Phosphine (Aluminum phosphide) is a commonly used fumigant in Pakistan. The fumigant applicators themselves reported chronic illness when in contact with the fumigant.2 Evidence from previous literature suggests phosphine gas (fumigant), which possesses a smell similar to that of garlic, is extremely hazardous to human health.3 It is commonly known as “suicide poison” which can be easily accessed and to date, has no particular effective antidote.1 There are very few reports of phosphine poisoning cases which clearly concludes congestion, pulmonary edema, and focal myocardial infiltration, to small-vessel injury as lethal effects of phosphine inhalation however all of the cases showed presence of phosphine in blood and liver specimens.4

There are no specific diagnostic tests developed to confirm the diagnosis of phosphine poisoning, however some previous studies focusing on phosphine poisoning
have found silver nitrate test and biochemical examination of the gastric aspirate as potential markers for its diagnosis but it is not available routinely hence the diagnosis is made on suspicious history and clinical suspicion.5-7

The objective of this study was to investigate various presentations of phosphine poisoning to ED in a low-income setting in Karachi, Pakistan. Through this study we tried to connect the dots on the extent, common presentations and reasons of exposure to phosphine poisoning.

CASE REPORT

After IRB approval, medical records of patients presenting to ED at Aga Khan University Hospital between January 2005 to September 2013 were reviewed to identify cases with confirmed or suspected diagnosis of phosphine poisoning. Details regarding exposure of phosphine were solicited from each patient/family. Other details pertaining to admission and management in ED and subsequent care were documented from the medical records. The nature of exposure and amount of phosphine was also noted.

There were seven cases, six were children and one adult female. All the patients reported to ED within 12-24 hours of beginning of symptoms, most of them presented with vomiting, abdominal pain, drowsiness and weakness. All had accidental exposure at home as insecticidal spray or salt placed in bedroom and had exposure with the fumes.

Case 1

Seven years old female patient weighing 18 kg presented with complains of generalized weakness, vomiting, abdominal pain and drowsiness followed by accidental exposure of fumigated phosphine at home two days prior to exposure and patient used this room as bedroom. There was no history of any other drug/poison ingestion or exposure. Other two adult family members were also exposed but were symptomless.

On examination she was cold and clammy, tachycardia and hypotensive, rest of the systemic examination was unremarkable.

She was managed with supplemental oxygen, fluids and inotropes support. Specific medication like magnesium sulphate and calcium gluconate was given.

Her initial laboratorial test were normal, CBC with raised TLC count, LFT and PT normal, cardiac troponin 0.7, serum electrolytes and creatinine levels were also normal. ABG revealed pH of 7.41, HCO₃⁻=12.9, PCO₂=20.4. ECG showed only sinus tachycardia, while her Echo was normal. She remained admitted for three days and then discharged safely.

Case 2

Thirty years old female patient weighing 55 kg presented to ED with restlessness and chest heaviness since 2 days along with shortness of breath and vomiting for one day, following exposure to the insecticidal tables placed in her bedroom 2 days prior to presentation, where she slept for 6-8 hours. Husband also exposed with same poison but for lesser period and was symptomless. There was no history of any other drug/poison ingestion.

On examination, she had unstable bradycardia, rest of the vitals and systemic examination was normal.

Lab Investigations were performed, here hemoglobin was 8.9 gm/dl, LFT was normal, APTT 20.7 sec, magnesium 1.6 and potassium of 3.1 which was corrected with oral potassium. Patient’s ECG was performed which showed sinus bradycardia and prolonged QT interval, with normal Echo. She was managed in ER with oxygen, and fluids. Injectable MgSO₄ was administered, while she remain on continuous ECG monitoring.

During six hours of ED stay, cardiologist consult sorted, she remained vitals stable other than bradycardia and rest of systemic examination was normal. Her chest discomfort settles, no more weakness, and difficulty in breathing or vomiting. However she was admitted in hospital for 6 days and was then discharged.

Case 3

Seven months old male patient presented with complains of fever, vomiting and drowsiness since last 4 hours. His mother revealed that last night they placed two insecticide tablets on his bed to kill the bugs and since morning he has these symptoms. No other history of any drug ingestion or poisoning was found and didn’t had any preceding illness.

On examination his vitals with respiratory rate of 40 bpm, heart rate of 140, blood pressure of 91/64 mmHg, Oxygen saturation 94% and was afebrile. He had no signs of meningeal irritation or abnormal CNS findings. His anterior fontanel in normal. Pupils were bilaterally equal and reactive. His CBC, electrolytes, creatinine and LFT were normal. ECG was also normal. Patients remained in ER for further management, and later was admitted in pediatric ward and was safely discharged after parents counseling and communicating the danger signs.

Case 4

Eleven years old female patient weighs 35 kg with similar types of exposure to phosphine presented with history of vomiting, abdominal pain, diarrhea, marked tachypnea and lethargy for last one day prior to admission. Two family members were also exposed to same poisoning but didn’t need consultation. Other than tachypnea (48/min), and bilateral wheezes and reduced air entry, systemic examination was unremarkable. She
remained in ER and was managed with fluids and supplemental oxygen. Her initial Blood pressure was 61/42 mmHg. Her CBC and LFT was normal. Serum, Creatinine and electrolytes was within normal range. ABGs showed respiratory acidosis. ECG revealed tachycardia of 146/min and short P-R interval, while CXR revealed ground glass appearance. Echo in ED revealed normal ventricular size, but reduce systolic function with a LV ejection fraction of 45% and dyskinetic LV motion. Because of tachypnea she was admitted in PICU, intubated and with inotropic support but expired within 12 hours of hospital admission due to severe RDS and non-responsive hypotension in spite of multiple inotropic support. His initial troponin was <0.5.

**Case 5**

Four years old female patient presented with the history of vomiting and abdominal pain for last ten hours. Family used insecticide tablets in their bed room she slept in.

On examination she was vitally stable but has garlic odor breath with no other systemic finding. Her lab results including CBC, LFT, electrolyte and creatinine were normal. ECG was also normal.

She remained admitted in the hospital for two days with symptomatic management and was later discharged safely.

**Case 6**

Eleven years old female presented to our ER after being exposed to phosphine gas at home via an insecticide, with history of vomiting and drowsiness for one day. On examination her vital was normal, same for her systemic examination.

She was admitted with IV fluids and supplemental oxygen. Her symptoms resolved in couple of days and was than discharged home.

**Case 7**

This five year male child had similar history of exposure to insecticide at home, in his bed room and presented with vomiting, diarrhea and drowsiness since 12 hours. Lab investigations were normal and was discharged after three days on admission to pediatric ward.

**DISCUSSION**

Seven patients with suspected phosphine poisoning were reviewed, most common presentation were GI symptoms of vomiting. Other presenting features include drowsiness, weakness and respiratory distress. Five patients were either admitted to ward or discharged, one patient expired and one patient was discharged as Left against medical advice. All the patients were exposed to phosphine accidently.

Unintentional inhalation of phosphine gas is rarely reported, however occupational exposure is occasionally seen. From target animals to human victims; previous studies state that phosphine gas is the most widely used fumigant against pests and a key element in food security worldwide.

In developing countries like Sri Lanka, poisoning is one of the leading top ten causes of hospital deaths. There was a dramatic increase in poisoning cases and deaths caused by suicidal ingestion in India during 1990s, particularly in sub-urban and rural parts of Northern India.5,11 Poisoning cases have also been reported in France, Turkey, Germany and Iran.12,13

Phosphine is produced when Aluminum phosphide (pesticide) reacts with air, water or gastric acid in turn inhibiting cellular metabolism leading to cellular hypoxia which results in clinical findings which may include headache, vomiting, cough, depressed mental status, pulmonary edema, hypotension, cardiac dysrhythmias, liver and kidney failure, and severe gastritis and unconsciousness similar to the symptoms of patients in our case series.2,3,14 Fatality in phosphine gas exposures is generally reported as being due to cardiac toxicity. When phosphine is inhaled, it reacts with moisture in the lungs to produce phosphoric acid, which has the potential to cause severe blistering and edema.2

Patients with phosphine poisoning present with non-specific clinical features. Two of similar studies including one conducted in North Iran shows nausea and vomiting as the most common presenting feature similar to our study with most of the patients improving on appropriate management and mortality rate of only 18.6%, as compared to our study with mortality rate of 14.3%.15,16

The initial toxicity of phosphine poisoning may be non-specific and transient; however in severe exposure patients develop metabolic acidosis, cardiovascular collapse, oliguria, and proteinuria eventually leading to anuria, which may require hemodialysis. Phosphine (Aluminum phosphide) in low doses has the potential to cause headache, dizziness, numbness, general fatigue, breathing difficulties (tightness around the chest, pain in the region of the diaphragm and cough) and gastrointestinal disturbances (pain, nausea, vomiting and diarrhea) while victims of chronic exposure exhibit lung irritation, persistent coughing, tremors and convulsions eventually leading to pulmonary edema, myocardial injury, kidney damage and coma. The cause of death varies from usually within the first few hours to up to two weeks in the case of liver failure or cardiovascular failure. Cardiac Arrhythmia and need of mechanical ventilation are considered as predictor of poor outcome.17

Phosphine is a highly toxic gas however the exact mechanism of action is yet to be known hence the management of cases with phosphine exposure includes supportive care as there is no accepted specific treatment.
Variety of possibilities available includes intravenous MgSO₄, N-acetylcysteine, pralidoxime, or trimetazidine, as well a combination of insulin and glucose administration along with hyperventilation as there is no antidote present as of today. Airway protection is of utmost importance along with cardiac support and monitoring until the phosphine is excreted out from the body, specific management of phosphate gas poisoning is to reduce its absorption and cellular toxicity and increase its excretion from the body.

This study showed all of the poisoning cases to be accidental rather than suicidal and were encountered after exposure to phosphine from insecticides most commonly bug sprays and tablets which were used a night prior without the knowledge of hazardous contents of the insecticide. Hence it is very important for people to be aware of the components of insecticides commonly used at home to prevent any accidental exposure leading to fatalities and industries to replace phosphine with an alternate compound which is safer and less hazardous for domestic use.

CONCLUSION

Phosphine compounds are cheap and easily available lethal poison with no available specific antidote. Exposure to phosphine is most commonly accidental via insecticide use locally leading to life threatening signs and symptoms and increases the risks of major morbidity and mortality. Hence there is a need to replace insecticides in a safer chemical compound and keep children out of reach from such toxins. Moreover increase awareness program and proper labeling and precautions should be written on all commonly sold pesticides and the constituent of hazardous compounds they contain.

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