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Phosphine Gas Poisoning in Children: A Case Series Study from Tertiary Health Care Center of Karachi, Pakistan

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CASE STUDY

ABSTRACT

Phosphine poisoning is an uncommon but hazardous public health issue. It is widely used as chemical insecticide and pesticide domestically and exposure to this chemical, mostly accidental among children, 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. We observed a short case series form a family who was exposed to phosphine gas from aluminum phosphide tablets that were used for fumigation purpose in bedroom. Family locked the house for six hours only and then used that bedroom without proper cleaning and removal of the chemical from the house. No adequate aeration of the room before use was done. Detail history from parents was taken and clinical details were identified however further details were noted from their medical records. We identified three cases of phosphine poisoning during this exposure. One of the babies was brought dead, with history of acute onset nausea and vomiting followed by increasing paleness, sweating and questionable seizure episode along with loss of body posture and unresponsiveness. Other two girls presented with generalized weakness, nausea, vomiting and lethargy since morning. Both sisters were admitted to pediatric intensive care unit for less than one week and were managed symptomatically along with magnesium sulfate. The objective of this case series study is to bring in attention of the families, health care community and the stake holders for the need to replace harmful insecticide fumigation practices with the safer ones, as phosphine gas can cause late onset of clinical feature and sudden death, probably because of fatal myocarditis as observed by echocardiography in both survived girls. Whenever fumigation is done parents/family members be provided with proper written guidelines that along with fumigation process and reuse of the house must mention possible side effects. Moreover, to increase public awareness, proper labeling and precautions should be written on all commonly sold pesticides and the constituent of hazardous compounds they contain.

INTRODUCTION

Phosphine (PH3) is a gas that is produced from a variety of chemicals that are usually used as

insecticides and rodenticides (1,2). The most common source of phosphine gas production leading to poisoning in human beings is produced from aluminum phosphide. Aluminum phosphide tablets are commonly used in houses and other living places to kill the insects and rodents, primary because of their low cost (3). Phosphine gas is produced when aluminum phosphide comes in contact with environmental moisture. It is a highly toxic, colorless gas having smell of putrid fish and garlic (4,5). Inhalation of phosphine gas leads to severe toxicity causing multi-organ failure including liver, heart, lungs and kidneys (6,7). Mortality reported from phosphine gas is quite high i.e. 30%-77% (8). Mechanism of multi-organ toxicity is involvement of the gas in multiple mitochondrial enzymes includes oxidation processes and free oxygen radical production (9). Till date no antidote is approved for phosphine gas poisoning but studies are going on boric acid and some studies have found it significantly useful (2).

MATERIAL AND METHOD

CASE SERIES

We are presenting case series of 3 siblings who were exposed to aluminum phosphide poisoning secondary to fumigation. Parents used aluminum phosphide tablets for fumigation and after around 6 hours they came back home and remained in that room overnight. From next morning their 3 daughters started having symptoms of vomiting, middle one developed symptoms first followed by younger one and finally elder one. Although parents were asymptomatic. One sister (middle one) was brought dead in emergency department and other 2 sisters landed in ED with vomiting. Younger sister was in sick condition and elder one was little bit stable condition and middle one was the one who was brought dead.

Case 1

Three years old female landed in pediatric ED with no breathing and no respiratory efforts. Patient was declared death on arrival. She was having history of vomiting since morning, that over time increased in severity, so that child was vomiting everything, she landed in ED dead. Parents were also giving history of progressively increasing pallor and sweating over the time and one episode of fit like activity before she became unresponsive. No workup was done and no treatment was given to her.

Case 2

One year and 4 months old girl weighing 8 kg presented with complaints of acute onset vomiting since morning, there were no associated symptoms, no fever, and no loose stools. On examination she was dull looking, dehydrated and tachypneic having acidotic breathing. Peripheries were cold and pulses were feeble, CRT was delayed but she was maintaining blood pressure (BP 90/53 mm-Hg) and saturation was 99% at room air. On systemic examination toddler was significantly tachypneic with otherwise clear chest. Other systemic examination was unremarkable. Initial work up sent in pediatric ED was remarkable with ABGs showing pH 7.46, PCO_2 24.20 mmHg, PO_2 93.10 mmHg, bicarbonate 16.90 mEq/L, base excess -4.9 mEq/L, O_2 sat 97.60%. CBC, LFT, electrolytes and PT/INR were normal and lactic acid was 2.2 mmol/L. Serum glucose level remained normal throughout course. Most significant lab findings were the cardiac enzymes that were significantly elevated. Initial trop I levels of patient were 0.131 ng/ml (lab reference range <0.04ng/ml) that gradually increased over succeeding day to 0.487 and 0.586 on second and fourth day of admission , respectively, before normalizing. Similarly, Pro BNP level was also significantly elevated 2719 pg/ml (lab reference range 125pg/ml) on first day and increasing to 9181 on second day before norming back to 119 on 4th day. Chest x ray done showed signs of pulmonary edema.

During ED stay patient also developed sinus arrhythmia and echocardiography done in ED showed mildly dilated left ventricle without hypertrophy and ejection fraction was 34 %. Patient was admitted to PICU and treated with high flow oxygen and magnesium sulfate, given every 6 hourly under strict monitoring. Double inotropic support was started i.e. epinephrine and milrinone along with diuretics and ACE inhibitors. Later on, when cardiac functions improved, patient was moved to general ward before discharge, with total hospital stay of 6 days.

Case 3

Four years old girl weighing 20 kg presented with same complains as her sisters since morning. She was the most stable among all 3 sisters and there were no other sign or symptoms except for vomiting. On arrival child was dull looking but conscious and oriented to time place and person. She was vitally stable with heart rate 104 /min, respiratory rate 26 /min, blood pressure 101/69 mm-hg, oxygen saturation 96 % and she was afebrile with temperature 37 ^{0}C. Systemic examination was also unremarkable. Patient blood gases done in ED showed pH 7.37, PCO_2 33.90 mmHg, PO_2 53.60 mmHg, bicarbonate 19.0 mEq/L, base excess -4.0 mEq/L, O_2 sat 99.70% and lactic acid 1.7 mmol/L. CBC, LFT, electrolytes, blood glucose and PT/INR were normal. Patients cardiac enzymes were also monitored. Trop I level on first day was 0.155 ng/ml (lab reference range <0.04 ng/ml) and the repeated result on second day was 0.0578. Pro BNP level on first day was 101 pg/ml (lab reference range 125pg/ml) on first day that contrary to Trop-I level increased on the second day to 595pg/ml. Chest x ray done showed signs of mild pulmonary edema. ECG done in ED showed sinus arrhythmia and echocardiography was done that turned out to be normal. Patient given IV fluids with magnesium sulfate and calcium gluconate along with potassium for cardiac stabilization and patient was admitted to in-patient facility in PICU for strict monitoring. During hospital stay patient was monitored strictly for arrhythmias and magnesium sulfate and calcium glucoronate treatment was continued. As patient remained vitally stable so was discharged in stable condition on 5th day.

RESULTS AND DISCUSSION

Accidental poisoning in children a global issue that has increased in last century even in developed countries due to expansion in pharmaceutical and chemical industry throughout the globe (10,11). Almost 300,000 people die each year due to pesticide and rodenticide poisoning (12). Aluminum phosphide (phosphine) is commonly used in Pakistan for fumigation purpose. Phosphine poisoning can present with variable symptoms and have a varied course. Initial symptoms are non-specific and may suggest a simple pathology like acute gastroenteritis. Initial symptoms include vomiting (usually having garlic odour), diarrhea, dizziness and epigastric pain (6,7,13).

All three of our patients were exposed to accidental phosphine gas. Aluminum phosphide tablets were used for fumigation purpose and family was completely unaware of the side effects that can occur. Family bought these tablets from over the counter from some vendor so they were not counseled properly regarding the duration of fumigation and safe withdrawal of the gases from the rooms. This led to the catastrophe that this family suffered. One middle sib landed in emergency in dead state and unexpectedly younger and elder sisters were carried to emergency latter on in unstable condition. Initially no diagnosis was made as parents were giving vague history of deceased baby but when 2 other sibs were brought in the ED with GI symptoms followed by respiratory distress, then direct questioning pertaining to the possible poisoning were asked. It then revealed out that they had their home fumigated for around 4-6 hours before they came back and started living in same home. In our study patients developed symptoms late. They were exposed to poison at around 8 pm on one day and symptoms appeared on the next morning, starting with

vomiting and followed by respiratory distress and death of one baby within few hours.

Although phosphine gas inhalation is uncommon but most of these exposures occur at occupational settings, unintentional inhalation is rarely reported in literature (14). It is still rare in children but due to its increasing use for preservative (widely used for wheat preservation) and fumigation purposes and its easy availability suicidal use of aluminum phosphide is increasing in many regions of the world (15).

Phosphine poisoning presents with a wide variety of symptoms that may match with some of the common infections as well. It usually starts with gastrointestinal symptoms like vomiting, stomach pain, loose stools followed by dizziness, lethargy, respiratory distress, arrhythmias, cardiac failure and ultimately leading to death, if not intervened at the proper time with the appropriate therapy (6). Phosphine poisoning leads to multi-organ involvement thereby causing pulmonary edema, liver failure, cardiac failure and renal failure. Most common cause of death in aluminum phosphide/phosphine poisoning is cardiac arrhythmias (like bradycardia, ventricular tachycardia etc.) and cardiac failure (13,16,17).

All three of our patients presented with history of vomiting followed by pallor, sweating and dizziness. Majority of the other studies report similar symptoms including Shadnia et. al. and Elabbassi et. al. (6,16). In our patients, cardiac enzymes were deranged and in one patient echo showed decreased ejection fraction and both patient developed arrhythmias although it was sinus arrhythmia. Akkaoui et. al. reported similar findings in their study in a case with myocardial injury secondary to phosphine poisoning (18).

CONCLUSION

Phosphine compounds are cheap and easily available lethal poisons with no available specific antidote. Exposure to phosphine is most commonly accidental via insecticide use leading to life threatening signs and symptoms and increases the risks of major morbidity and mortality. Hence, there is a need to replace insecticides with a safer chemical compound and keep children out of the 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.

RECOMMENDATIONS

We recommend that a high index of suspicion for poisoning should always be given to any child who presents with unusual findings and whenever 2 or more family members present with the similar or related complains. Whenever phosphine/aluminum phosphide poisoning is suspected patient must be referred promptly to the health care facility where intensive care facility is available along with multi-disciplinary team, so that these patients can be managed properly, as only treatment is supportive in many of the poisonings including phosphine gas poisoning.

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