



Rice tablet: An overview to common material in Iran

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Article info

Article History:

Received: 29 Mar 2016

Accepted: 24 Apr 2016

ePublished: 31 May 2016

Keywords:

Aluminium Phosphide,

Rice Tablet,

Toxicity

Abstract

Rice tablets or aluminum phosphide (ALP) poisoning is one of the most lethal poisoning cases in the developing countries, leading to high mortality, especially in youths every year. The management of ALP poisoning remains purely supportive because no specific antidote exists. There are various sign and symptoms that can occur due to ALP consumption. The main way to a diagnosis of the ALP consumption is history. Early diagnosis can rescue the patient; thus, the diagnosis and supportive treatment is so important.

Citation: Amiri H, Vaseie L, Habibollahi P, Ghodrati N, Hoseini-Kasnavieh SM. **Rice tablet: An overview to common material in Iran.** J Anal Res Clin Med 2016; 4(2): 77-81. Doi: 10.15171/jarcm.2016.013

Introduction

Rice tablets or aluminum phosphide (ALP) poisoning is one of the most lethal poisoning cases in the developing countries, leading to high mortality, especially in youths every year. The management of ALP poisoning remains purely supportive because no specific antidote exists. Although in various studies mortality rates have been reported ranged 40-80%. In many cases, it marked too late, suspected to consume of botanical rice tablets (garlic compound) and caused delay in aggressive treatment. ALP is an effective insecticide and rodenticide, which is used widely in the storage place and transportation of grain as a fumigant to control rodents and pests. For many years, ALP and zinc phosphide have been strong insecticides and rodenticides, which are

cheap and effective, and their residues are not toxic.¹⁻⁴

Due to the high probability of death from poisoning by ALP, early diagnosis and intervention action can prevent interact with water and release phosphine gas is the best action to save the patients. When start working, the only possible way is to control symptoms and symptomatic treatment.⁵

Mechanism of Action

Phosphide combined with atmospheric moisture and released two or more phosphine hydride PH₃, which is an active ingredient of poison.



Toxicity is caused by the liberation of phosphine gas when phosphide interacts

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with the moisture present in the air between grains or when ingested phosphides come into contact with gastric fluid.

Liberation of phosphine happens more in the acidic environment. Acute poisoning with these substances is divided into three categories: (1) direct swallowing, (2) breathing air contain phosphine, and (3) rarely absorbed through the skin.

Pure phosphine is colorless and odorless. However, phosphine contains impurities that are responsible for the garlic-like smell that can be detected when phosphine concentrations are as low as 0.02 ppm.

A potential mechanism for this material (phosphine) is cytochrome oxidase blocks and inhibition of oxidative phosphorylation and finally cell death. So, organs such as the brain, heart, liver, and kidney that require more oxygen than the amount of the toxin are more sensitive and more vulnerable.

Hence, to the production of free radicals in several tissues by phosphine, those organs, which need more oxygen, such as heart, brain, lungs, kidney, and liver have more sensitivity to phosphine-related damages, and this is in association with histopathological changes in those organs.⁶⁻⁹

Complications

Acute: Inhalation of phosphine cause a headache, dizziness, fatigue, cough with sputum (green) moving difficulty and speaking, nausea and vomiting, gastritis, and diarrhea.⁴

Oral consumption

Nausea, vomiting, diarrhea, stomach pain, shortness of breath, chills, headache, convulsions, and coma.⁴

Chronic inhalation

Appetite loss, weight loss, anemia, spontaneous fractures of bones, teeth ache and mandibular swelling, and necrosis. The effective ingredient of toxicity in these patients is phosphine gas, PH₃, which absorbed through the mucosal tissues after release in the gastrointestinal tract and distributed,

especially the heart and lung tissues and vascular. This hypothesis is further confirmed by the presence of phosphine in the blood circulation of death patients.⁴⁻¹⁰

Changes in cell morphology, lipid peroxidation, inhibition of catalyze and peroxides and inhibition of cholinesterase are its consequences. Created cell hypoxia and impact of toxin on production of catalase and cell peroxides reduce capacity of collecting harmful radicals peroxides and resulting cell damage.^{11,12}

It seems that one of the important ways to transfer phosphine is connecting with the red blood cells, which in some cases lead to hemolysis or methemoglobinemia. Cause of death in these patients is resistant to treatment which may happen vasogenic shock or cardiogenic shock and usually happens in extreme acidemia.¹³

Clinical signs

There are several studies about cardiac involvement that indicate electrocardiographic (ECG) abnormalities and decrease cardiac output, congestion, and edema of most of the organs.

Echocardiography of these patients have been reporting increase of size, hypokinesia, and dyskinesia of left ventricle and decrease cardiac output as much as $36 \pm 9\%$.¹⁴⁻¹⁸ They resolve when patients rescue.

Use of echo in death patients indicates an increase of size of the left ventricle in the 1st day in compared with those who survived the 5th day. It has shown hypokinesia and slow motion of left ventricular septal and in some cases akinesia, which they improved over time and usually until to the 4th day in survive of the patient. There are any reports about survival of patients after cardiogenic shock.¹⁴

In autopsy of these patients, edema of myocardial fibers, heart congestion, vacuolization and focal necrosis of myosites, and myocardial cell infiltration are significant findings. Although in all death patients especially who death earlier than 12 hours was not observed. ECG abnormalities were seen in

38% of cases.¹⁹ The most common patient ECG changes in ST segment and T-wave.

Tachycardia and bradycardia and in all types of arrhythmias have been reported on the toxicity.^{15,19,20} No significant correlation between the types of cardiac arrhythmias and outcome of this disease have not earned.

Totally, cardiovascular symptoms of patients include severe hypertension, reduced cardiac output, reduced vascular contractility, increase of systemic venous pressure, and pulmonary artery wedge pressure.^{14,19}

Pulmonary symptoms of patients are including pulmonary edema, Ronkei, tachypnea, dyspnea, and crepitation. Pulmonary edema is common within 4-8 hours after poisoning, and its pulmonary or cardiac origin is not clear. Normal pulmonary artery wedge pressure with loss of saturation can be a cause of non-cardiac edema. Some studies have reported acute respiratory distress syndrome.^{6,15,18,21-23}

Gastrointestinal symptoms including blood vomiting, erosions of the stomach and duodenum,^{24,25} and dysphagia in alive patients esophageal fistula stenosis have been reported. The increase of aminotransferase and with less prevalence is possible. Disseminated intravascular coagulation symptoms of hemolytic anemia Heinz bodies can be named.²⁶

Electrolyte imbalance including hypokalemia, metabolic acidosis with alkalosis respiratory, and kidney failure are common. Hyper- and hypo-glycemia, hyper- and hypo-magnesium can be symptoms of poisoning.⁶ Hypoglycemia can be prolonged and severe.²⁷

Uncommon presentations are pleural effusion, ascites, rhabdomyolysis, tendency to bleeding (bleeding diathesis), kidney failure (acute tubular necrosis) pericarditis, methemoglobinemia, and microangiopathic hemolytic anemia.⁶

Diagnosis

The study showed that using silver nitrate test 1.0 normal from gastric secretions can be

proved 100% of cases and by through breathing 50% of cases presence of phosphine. Some studies have reported diagnoses based on clinical suspicion and oral contraceptive use by patients or caregivers.²⁸⁻³⁰

Treatment

Treatment is in common shape is maintenance because of its specific antidote.^{6,31} In the normal treatment of these patients, gastric lavage treatment should be avoided because of the phosphine gas. There is little evidence for the use of charcoal.³² In laboratories environment, vegetable oils and liquid paraffin are barrier for phosphine release.³³ However, it has not been tested in the clinic. Although coconut oil in the early evacuation of the stomach and sodium bicarbonate and activated charcoal is used orally to patients.

There is not experimental study for bicarbonate use, but it seems it does not reduce mortality and morbidity. Despite the lack of empirical studies diluted potassium permanganate is also recommended.³⁴ Some studies have found it without benefit^{35,36} and some have found useful.^{37,38}

Although glutathione and the activity of acetylcholinesterase, the blood is reduced. The treatment role of N-acetyl cysteine and pralidoxime treatment is not clear in the clinic, and more studies are needed to prove their usefulness.³⁸ The use of hyper insulin protocol-euglycemia and hyperventilation with the entry of calcium into heart cells improve cardiac contraction.³⁹ In a study conducted by Modi et al.⁴⁰ indicated that there is no direct relationship between the number of pills and death.

Six important prognostic criteria include pH < 7.2, bicarbonate < 15, lower Acute Physiologic Assessment and Chronic Health Evaluation (APACHE) score, early mechanical ventilation necessity, need for premature mechanical ventilation, vasoactive and vasopressor and increase of creatinine.³⁹

Conclusion

Due to the very high probability of death

from poisoning by ALP, early detection and short interval between consumption of this tablet and the immediate start of treatment can be the important factors to prevent the early death in intoxication with this tablet.

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Conflict of Interests

Authors have no conflict of interest.

Acknowledgments

None.

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