دانشگاه علوم پزشکی و خدمات بهداشتی درمانی
استان کهگیلویه و بویراحمد
معاونت غذا و دارو

In the name of GOD

Zinc and aluminum phosphide poisening

- Commonly found in powder, pellet, or tablet form, the metallic phosphides, zinc and aluminum phosphide, are both low-cost and highly toxic rodenticides.

- Each tablet contains 3 grams of aluminum phosphide, which releases 1 gram of phosphine gas per tablet. Ingesting a quarter of a fresh tablet can be fatal and cause death.

- Inhalation of phosphine gas, produced when aluminum or zinc phosphide is exposed to moisture in stored grain, represents the most common form of exposure.

- When the tablet is exposed to moisture or stomach acid, phosphine gas is released. Phosphine gas is a potent protoplasmic poison that interferes with the activity of enzymes and intracellular proteins.

mechanisms of toxicity:

- inhibition of oxidative phosphorylation
- free radical production with promotion of lipid peroxidation
- cholinesterase inhibition
- Decrease glutation
- cause circulatory collapse
- Cause fluid loss
- Cause adrenal damage

Risk factors for death after intentional poisoning include:

- dose (≥500 mg of phosphides)
- hypotension
- acidosis
- hypoxia
- global left ventricular hypokinesis, and left ventricular ejection fraction <40 percent

- Ingestion of fresh phosphide rodenticide in the original packaging is most potent.

- phosphine gas is known to have an odor similar to rotten fish and is detectable to some at a concentration of 2 ppm, it is not a reliable early warning sign of exposure.

- Mortality often occurs rapidly within the first day of severe metallic phosphide poisoning regardless of therapy.

- Death typically results from cardiac arrhythmias or refractory shock and cardiac failure.

Clinical manifestations and diagnosis

- Gastrointestinal (GI) irritation marked by nausea, vomiting, hematemesis, and retrosternal chest and abdominal pain

- Shock with refractory hypotension caused by direct cardiac toxicity

- Cardiac arrhythmias, including bradycardia, supraventricular tachycardia, atrial fibrillation, atrial flutter, and ventricular arrhythmias

- Hemorrhagic pulmonary edema with tachypnea, cough, acute respiratory distress syndrome, and respiratory failure

- Less common features include hepatotoxicity, intravascular hemolysis with methemoglobinemia and/or renal failure
- Hyperglycemia: poor prognosis
- Abnormal electrolyte

DIAGNOSIS:

- The diagnosis of phosphide poisoning is made by history and characteristic clinical signs. (hypokalemia and elevated lactate concentrations may be seen)

- zinc phosphide is radiopaque

- Phosphine may be released as a gas from emesis, feces, or lavage material and can cause respiratory distress in health care providers and other exposed persons.

- However, serious toxicity in health care providers caring for patients poisoned with metallic phosphides has not been described

TREATMENT:

- For phosphide ingestion, supportive care is the mainstay of treatment and consists of the following :

- Provide supplemental oxygen and ventilation as needed and dictated by the degree of respiratory compromise. Tracheal intubation may be performed in standard fashion.

- Gastrointestinal decontamination

- Provide fluid resuscitation with rapid infusions of isotonic normal saline to replace obvious fluid losses and to treat hypovolemic shock treat hypoglycemia and correct hypokalemia an hypomagnesemia as indicated.

- Treat cardiogenic shock with vasoactive medications as needed in patients unresponsive to isotonic fluid resuscitation

- magnesium infusion appears to be of greatest potential benefit.

Adjunct therapies include:

- Magnesium infusion

Hypomagnesemia should be corrected in all patients with metallic phosphide poisoning

Small trials suggest intravenous magnesium administration can decrease mortality,

- the regimen with the best effect was as follows:

1 g magnesium sulfate, intravenously followed one hour later by 1 g given as a continuous infusion over three hours and then 1 g every six hours until recovery or a maximum duration of five days.

- Insulin and dextrose infusion:

Thus, this therapy may be beneficial in patients who are not responding to supportive care and who are unlikely to benefit from magnesium infusion.

A hemodynamic response to high-dose insulin therapy is delayed for 30 to 60 minutes, therefore simultaneous implementation of other therapies to support the patient's pulse and blood pressure are generally required.

- Repletion of potassium and magnesium may be needed.
- Serum glucose, serum potassium, and fluid intake and output should all be closely monitored for the duration of treatment with high- dose insulin.
- We suggest measuring glucose (eg, fingerstick) every 15 to 30 minutes while titrating the insulin infusion rate, and approximately every one to two hours once a steady rate is determined and glucose measurements are stable.

- For patients with a serum glucose concentration below 150 mg/dL (8.25 mmol/L), we administer 50 mL of 50 percent dextrose (D50W) IV.

- For patients with a serum potassium concentration below 3 mEq/L (3
- mmol/L) , we administer 20 mEq of potassium IV.

- We initiate high-dose insulin therapy with a bolus of 1 unit/kg of regular, short-acting insulin given IV. Following this bolus, we begin a continuous infusion at 0.5 units/kg per hour IV and titrate upwards until hypotension is corrected or a maximum dose of 10 units/kg per hour is reached.

- One approach is to increase the infusion rate by **50 percent every 20 minutes** until either target is met.

Other therapies:

- Individual case reports describe the use of N-acetylcysteine (NAC) as an antioxidant and the antianginal agent trimetazidine to maintain oxidative phosphorylation.

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سپاس از توجه