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“ zinc phosphide poisoning From A To Z ”

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ABSTRACT:

Toxicity from rodenticides as metal phosphides is common in the whole world, Suicidal behaviors are the most frequent cause of poisoning, followed by unintentional exposure. The average age is close to 21.

Zinc phosphide is a Gray crystalline substance or gray black powder. it is a popular domestic rodenticide due to its accessibility, low cost and high availability.

Phosphides become hazardous quickly after consumption, usually within 30 minutes.

It enters the body and is transformed into phosphine gas upon consumption. Phosphine works by inhibiting cytochrome oxidase, which impairs mitochondrial function. Apart from causing cell energy failure, free radical, which also causes energy failure in cells

Metal phosphide poisoning results in cyanosis, hypotension, shock, palpitations, nausea, restlessness, stomach discomfort, and cardiac arrhythmias, Hepatitis, acute tubular necrosis, disseminated intravascular coagulation, and pulmonary alkalosis are additional uncommon side effects.

Since there is sadly no known antidote to lower the high death rate, it is crucial to identify these patients and treat them as soon as possible.

Conclusion: ZnP can result in potentially fatal consequences and is the active ingredient in many readily available insecticides.It is critical that these patients are identified and treated as soon as possible. Regretfully, there isn't a particular antidote that could lower the elevated patient death rate. Initial symptoms are nausea, agitation, palpitations, pulmonary edema, cyanosis, hypotension, shock, and cardiac arrhythmias. Understanding its pathophysiological foundations enables a suitable approach that, when combined with existing treatment strategies, not only increases survival but also provides a means of conducting additional research into the mechanisms of action of individual therapeutic resources as well as the creation of more effective therapeutic alternatives.

Key words: zinc phosphide , poisoning , metal , Rodenticides.

Introduction

Across the world, rodenticides like metal phosphides can be toxic. This is especially true in underdeveloped nations where people have access to unlabeled and unregulated pesticides and insecticides. Many people have employed zinc phosphide as a rodenticide.[1]

Zinc phosphide is a rodenticide that has been used since 1940 in agricultural, urban and industrial environments . Since 1985, there are specific restrictions in the Regulations, Guidelines and Standards on its use as a pesticide.

It is even prohibited in several countries. In North America, zinc phosphide is not prohibited or restricted for importation, manufacture, formulation, commercialization or use .This makes it accessible to the general population, and although a cause–effect relationship between its availability and its use for suicidal purposes has not been reported, its accessibility makes it an easy xenobiotic to obtain, regardless of the purpose for which it is used.[10]

Suicidal attempts are the most frequent cause of poisoning, followed by unintentional exposure. The average age is close to 21. The most frequent causes of self-poisoning include marital discord, financial difficulty, social issues, and criticism from other family members. [1]

Zinc phosphide Gray crystalline substance or gray black powder. Because zinc phosphide (Zn_3P_2) is highly available and inexpensive, it is frequently used as a rodenticide in homes..[2]

Coloring rodent baits helps people distinguish toxic baits from food or feed, thus preventing accidents caused by human errors .

In order to avoid cases of primary poisoning by children, AR for amateurs should be placed on the market on reclosable or non-reclosable .

Reclosable packages Child-resistant fastenings used on reclosable packages shall comply with ISO standard as amended relating to ‘Child-resistant packages Requirements and methods of testing for reclosable packages’ adopted by the European Committee for standardisation (CEN) and the International Standard Organisation (ISO). [11]

Black powder of zinc phosphide: figure 12

Mechanism of toxicity

Phosphides can cause poisoning very quickly—usually within 30 minutes of consumption—and can cause death in less than six hours. When new, unopened tablets are consumed, the outcome is always fatal. Ingesting more than 500 mg of phosphorus can be lethal.

It enters the body and is transformed into phosphine gas upon consumption. Following its absorption into the bloodstream via the stomach and intestines, phosphorus is subsequently absorbed by the liver and lungs. Numerous harmful consequences, both metabolic and nonmetabolic, are caused by phosphorus gas. [3]

Phosphine works by inhibiting cytochrome oxidase, which impairs mitochondrial function. Lipid peroxidation is caused by an increase in free radical formation, which also causes energy failure in cells. In rats, phosphorus also inhibits cholinesterase.[1]

Figure (2): zinc phosphide[13]

It is also known that phosphorus inhibits enzyme activity and protein synthesis, especially in the mitochondria of heart and lung cells. The electron transport chain in the mitochondria may get blocked as a result. Moreover, it might denaturize a number of the enzymes necessary for cellular metabolism and respiration. It is phosphine that causes denaturation of oxyhemoglobin molecule.

Oxyhemoglobin is gradually changed into methemoglobin and hemichrom species by it. Phosphite and phosphate are produced from reaction between oxyhemoglobin and phosphine ions Phosphine also, minimizes the amount of oxyhemoglobin of blood . Phosphine then causes brain, lung and liver oxidative damage .[4]

Clinical picture

Metal phosphide poisoning results in cyanosis, hypotension, shock, palpitations, nausea, restlessness, stomach discomfort, and cardiac arrhythmias.Hepatitis, acute tubular necrosis, disseminated intravascular coagulation, and pulmonary alkalosis are additional uncommon side effects. Phosphine gas inhalation results in delirium, convulsions, tremors, cold sweats, pulmonary edema, coma, and death from cardiac and respiratory arrest. Deep circulatory collapse, the primary fatal result of phosphide consumption, is related to causes such as injury to the adrenal glands, fluid loss, and direct effects on cardiac myocytes.Moreover, phosphine and phosphides have corrosive action corrosively. [3]

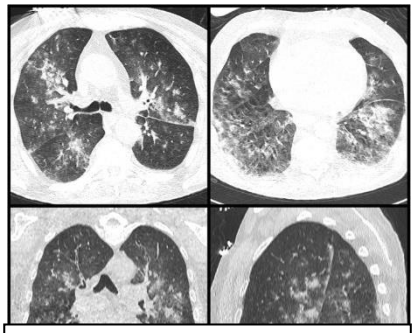

Figure (3) : CT showing Alveolar atelectasis from base to to apex [14]

Ocupational exposure to phosphine causes epigastric pain, GIT upset, headache, cough, tightness around the chest , giddiness, numbness, lethargy, anorexia and. The abnormal physical signs included bilateral diffuse rhonchi and absence of ankle reflex.[4]

The time between phosphide administration and the onset of systemic toxicity is typically quite brief. Circulatory failure is caused by phosphorus-induced decrease of cardiac contractility and fluid loss. Crucially, pulmonary edema ensues, albeit it is not always evident if this is a cardiogenic or non-cardiogenic condition. Acute renal failure and metabolic acidosis, or a combination of respiratory alkalosis and metabolic acidosis, are common. Hepatic necrosis, renal failure, and disseminated intravascular coagulation are other features .[5]

Management

Therefore, early gastric lavage along with the oral administration of sodium bicarbonate, activated charcoal, virgin olive oil, and intravenous magnesium administration are measures of potential benefits. It is recommended to apply gastric lavage frequently and with a lot of fluid.

Virgin olive oil is used to assist the treatment plan. It is crucial to administer low dosages of virgin olive oil at intervals to minimize the possibility of vomiting from the oil. It is best to begin therapy and diagnosis as soon as feasible.[6]

Metal phosphide poisoning currently has no recognized antidote, while several treatments, including hemodialysis, tranexamic acid, coconut oil, and castor oil, have been mentioned in case reports. Recuperation requires adequate supportive therapy, which is frequently provided in an intensive care unit.

Alpha-lipoic acid (ALA) is a naturally occurring coenzyme and antioxidant that has been used medicinally to treat alcohol- and diabetes-induced liver cirrhosis. However, a wide number of other medical disorders, including as lead poisoning and Amanita virosa toxicity, have been researched in relation to the potentially therapeutic benefits of ALA. . By acting as a glutathione (GSH) precursor or restorer, NAC demonstrated to reduce organ toxicity, particularly hepatotoxicity. It has also showed promise in treating ZnP poisoning . In two different studies , patients received a fixed combined dose of ALA, NAC, silymarin, and selenium. According to the studies, the therapeutic combined dose was proven to be helpful to individuals with viral hepatitis and alcoholism, significantly reducing liver function parameters without causing any noteworthy side effects. [7]

If more oxygen is needed, it's doubtful that additional steps to maintain airway control will be needed. bolstering care Even with intensive care, many patients will still die from metal phosphide poisoning. All that can be provided are supportive measures, which ought to be carried out in accordance with clinical developments. In every situation, a blood glucose level should be taken and, if hypoglycemia is discovered, addressed. Similar to this, hypokalemia should be sought for and, if clinically indicated, at least partially rectified; in some individuals, cardiac characteristics have disappeared when potassium concentrations were adjusted .

But keep in mind that life-threatening hyperkalemia can result from beginning of acidosis, renal failure, and cell damage. Conventional therapy should be used to treat metabolic acidosis. Supplementing with magnesium ,The difficult choice is whether or not to administer more magnesium. A course of treatment like this would seem nonsensical if magnesium depletion did not occur, but there have been isolated reports of situations when magnesium injection appeared to stop atrial fibrillation, SVT, and VT. However, although returning a normal magnesium content, 3 g of magnesium sulphate administered intravenously over a 30-minute period could not eliminate bigeminy or extremely frequent ventricular ectopic beats.

Pralidoxime

Acetylcholinesterase is inhibited by phosphine, as demonstrated by both clinical and experimental evidence. Studies examining the advantages of giving rats dosed with phosphide 10 mg/kg (5.55 times LD50) 5 minutes earlier atropine 1 mg/kg and pralidoxime 5 mg/kg parenterally. Nine of the fifteen animals had a 2.5-fold increase in survival time after receiving treatment, and the six remaining animals survived. The two control groups contained no survivors. Additional research is necessary to validate the advantages of oximes.[8]

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Case of zinc phosphide poisoning:

A 45-year-old woman with a history of untreated depression was brought to the emergency room 6.5 hours after consuming 6 to 9 g of zinc phosphide (weight dose calculated at 80–120 mg/kg). She was discovered at home in a supine position, oblivious to her surroundings, exhibiting involuntary uresis and black content emesis. Beside her was a 50 g vial containing 24% zinc phosphide. She then said that she had drunk 500 cc of beer and, afterward, three-quarters of the zinc phosphide bottle. She was awake when admitted to the emergency room and complained of anxiousness, nausea, and vomiting as well as epigastric pain. The following were her vital signs: 36.5 °C, or 129 beats per minute for heart rate, 94/74 mmHg for blood pressure, 87% SaO2 (FiO2 21%), and a 16-point FOUR scale score. Without delay, the clinical toxicological services were informed. An ECG

revealed sinus tachycardia, arterial blood gases with pH 7.298, pCO2 22.2, pO2 25.3, methemoglobin (MetHb) 1.1, carboxyhemoglobin (COHb) 0.6, HCO3 10.5, EB −14.3, and lactate 7.7. Upon admission (7 hours after intake), gastric lavage was carried out using 200 mL of coconut oil and 50 mL of 7.5% bicarbonate. The following procedures were carried out: injection of crystalloid solutions, request for laboratory testing, and continuous heart monitoring. She did exhibit a large anion gap and metabolic acidosis, which grew worse over the first six hours The hyperinsulinemia–euglycemia treatment was started eleven hours after the xenobiotic was consumed. It required doses up to seven IU/kg/h and a glucose contribution determined at 0.5 g/kg/h. The therapy lasted for 78 hours. The loading dose was 1 IU/kg/h and the maintenance dose was 1 IU/kg/h. The supply of serum glucose was cut off 21 hours after the insulin was stopped. Additionally, 1 g of magnesium sulfate was given every hour for 24 hours, and subsequently 1 g every 6 hours for 4 days. For seventeen treatments, N-acetylcysteine was administered orally, at a starting dose of 140 mg/kg and a maintenance dose of 70 mg/kg. always diluted in saline solution and 7.5% sodium bicarbonate with a 1:1 ratio. Treatment was provided with 20% lipid emulsion, 13 hours after the toxin was consumed, with a 1.5 loading dose mL/kg, a maintenance dose of 10 mL/h for 24 h and a therapeutic duration of 84 h. Additionally, a proton pump inhibitor, an antiemetic, and low-flow oxygen treatment were added hydroelectrolytic disease treatment and observation . She began to demonstrate clinical and biochemical improvement nine hours after the treatment began, and sixteen hours after that ,there was no Hemodynamic instability or tissue hypoperfusion. (without necessitating the need of bicarbonate replacement or vasopressor support). Seven days later, she was released without incident, with psychiatry and psychology providing follow-up care.[9]

Figure (4) : Residual sample (50 milliliters) of the diluted rodenticide ingested by patient.[15]

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