

A Case Report On Rat Kill Poisoning (Zinc Phosphide) In Rural Hospital At Wardha.

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ABSTRACT:

Background: A common rodenticide used against small animals is rat kill poisoning. It contains at least 32 percent zinc phosphide, which is extremely harmful to people when exposed to it quickly. It can be ingested mistakenly or on purpose. It enters the body through the gastrointestinal, respiratory, and dermal pathways. A respiratory toxin called phosphonate inhibits the cytochrome C oxidase pathway, causing renal and liver failure.

Case presentation: A 70-year old male patient was brought to the casualty by relatives in an unconscious state with a history of consumption of rat kill poison (zinc phosphide, of an unknown quantity, at his home in his village under the influence of alcohol. The patient had two episodes of vomiting following consumption in the hospital; it was not associated with blood. No history of giddiness, no history of loss of consciousness or seizures, no history of bleeding tendencies. Patient was a chronic alcoholic since 30 years; the last intake was today morning. He suffered from acute tubular necrosis and severe acute renal dysfunction. He had hemodialysis three times for tubulointerstitial nephritis and other resuscitation techniques. Over the course of three weeks, supportive counselling helped him to recover.

Conclusion: The harmful effects of rat poisoning, a widely used rodenticide, are mediated through the conversion of phosphide to phosphine gas. Refractory hypotension and arrhythmias were the main causes of the majority of deaths, which happened in the first 12 to 24 hours. With repeated hemodialysis, the patient in the present case started to improve. The majority of peripheral hospitals' lack of supportive care and renal replacement options makes it extremely difficult to offer prompt interventions to stop mortality.

Keywords: Rat kill poisoning, zinc phosphide, kidney injury, hemodialysis.

INTRODUCTION:

Consumption incidents of rat poison are among the second-most common poisonings in developing countries. Significant mortality and morbidity are linked to it. Some of the situations, nevertheless, are resolved with no consequences. This is a result of the content's diversity. Therefore, the mortality and morbidity of the poison used to kill rats depend on its chemical composition¹⁻³.

In India, zinc phosphide is an extensively used rodenticide to control the population of rats, mice, dogs, and ground squirrels. Depending on the application techniques, zinc phosphide is designated as a food ingredient or non-food chemical for use indoors and outdoors. Zinc phosphide is a food additive that is used on sugarcane, grapes, grasses, and in some areas, artichokes and sugar beets. As rodenticides, non-food items are utilized in indoor and outdoor residential, commercial, and agricultural areas. Due to its qualities of eating efficacy, safety, and dangers, zinc phosphide has a long history of usage as rodenticide. It is frequently utilized for rodent control on a global scale⁴⁻⁶.

In the United States, zinc phosphide was originally approved for use as a pesticide in 1947. In June 1982, EPA published a registration standard for zinc phosphate (PB85-102499). ADATA-call-In Notices (DCI) requesting further data for registration² were issued in 1982 and 1991, respectively⁷.

Phosphine gas and zinc hydroxide are produced when zinc phosphate combines with stomach acid and water. Since humans have emetic sensors and this phosphine gas has an emetic quality, people are the only ones who can emit it; rodents are unable to experience emesis. Phosphine inhibits cytochrome C oxidase after absorption in the GI tract, which is necessary for mitochondrial respiration and reduces cell energy production⁸⁻¹⁰. Weakness, anaemia, toothaches, necrosis of the jawbone, loss of weight, vision loss, and unconsciousness are signs of zinc phosphide toxicity. Under the brand names Arrex, Commando, Denkerin, Grains, Phosovin, Zinc Tox, Ratil, Rattle, and Rate Shot, among others, zinc phosphide is sold in India. We are finding that people are using zinc phosphide as homicide agents in the current period.

CASE PRESENTATION:

We present a case of a 70-year-old male who was admitted to the I.C.U. of the rural hospital Wardha. Patient was brought to the casualty by relatives in a conscious state with a history of consumption of rat kill poison (zinc phosphide), of an unknown quantity, at his home in his village under the influence of alcohol. Patient had two episodes of vomiting following consumption in the hospital; it was not associated with blood. No history of giddiness, no history of loss of consciousness or seizure. No record of bleeding, tenderness, chest pain, palpitation, breathlessness, abdominal pain, loose stools, blood in stools, no history of fever, cough and cold.

The patient has been a chronic alcoholic since 30 years; the last intake was today morning

no history of hypertension, diabetes mellitus, tuberculosis, or bronchial asthma. Pulse rate: 74, resp: 16, B.P: 90/60 mmhg. Temperature: afebrile. All routine investigations were done, with the following findings: **CBC investigations on cell counter with PS:** 12.1% haemoglobin, 4.23 red blood cells, and 4500 white blood cells, total platelets and 1.63. In the kidney function test, the values for urea were 80 mg/dl, creatinine was 3.5 mg/dl, sodium level was 150 mEq/l, and potassium was 5.5 meq/l. He developed severe acute kidney injury with acute tubular necrosis. Tubulointerstitial nephritis, for which he underwent hemodialysis three times along with other measures of resuscitation.

THERAPEUTIC TREATMENT IN HOSPITAL:

Gastric lavage was given at a time, Inj. Pan40 mg IV OD, Inj. Emset 4mg IV TDS. Inj. Vit. K 10 mg IV OD for 3 days. Inj. Mucomix 600 mg IV TDS. Inj. Optineuron 1amp in 100ml NS IV TDS, Inj. Thiamine 1ampoule in 100 ml NS IV TDS, Inj. D25 IV TDS. On discharge: Tab. Pan-D OD for days, Tab. Neurobion forte O.D. for 15 days, Tab. Benalgis OD for 15 days. Follow up in medicine O.P.D. after 15 days or SOS. He improved with supportive therapy over a period of 3 weeks. Daily input output monitoring, BP monitoring and RBS monitoring were done. Intravenous fluid was given to maintain the electrolyte balance. Vital parameters are being monitored every 2 hours. Keep the intake and output chart current. The patient was hemodynamically stable and hence was being discharged.

DISCUSSION:

Here we present a 70-year-old male patient who was brought to the casualty by relatives in an unconscious state with a history of consumption of rat kill poison (zinc phosphide, of an unknown quantity, at his home in his village under the influence of alcohol. The patient had two episodes of vomiting following consumption in the hospital; it was not associated with blood. No history of giddiness, no history of loss of consciousness or seizures, no history of bleeding tendencies. The patient has been a chronic alcoholic since 30 years; his last intake was today morning. He suffered from acute tubular necrosis and severe acute renal dysfunction. He had hemodialysis three times for tubulointerstitial nephritis and other resuscitation techniques. Over the course of three weeks, the supportive treatment helped him to recover.

Acute poisoning-related morbidity and mortality are a global problem with major medical, legal, and social consequences. In the final 25 years of the 20th century, industrial technologies, pharmaceuticals, and agriculture all made significant strides. These developments have coincided with modifications in acute poisoning trends in both developing and developed nations². One of the first methods used to attempt or commit suicide is self-poisoning. There are reports available from various regions of the world highlighting the toxicity and abuse of different chemicals for acute poisoning. India is a largely rural country with an agricultural economy¹¹⁻¹⁴.

Despite occasionally being used in public for suicide, zinc phosphide is frequently used as a rodenticide. In some parts of Asia, acute rodenticide self-poisoning is a serious issue. The most common way that humans are exposed to zinc phosphide, either accidentally or on purpose, is by ingestion. Although phosphine is responsible for most acute symptoms, unreacted zinc phosphide damages the liver and kidneys. It is easily absorbed through the gastrointestinal tract. Humans who consume zinc phosphide may get emesis¹⁵⁻¹⁸.

This case report mentioned that early signs of poisoning include nausea, vomiting, abdominal discomfort, tightness in the chest, agitation, and cold sensations. Treatment requires gastric lavage or inducing emesis to remove ingested zinc phosphide in a hospital setting. Activated charcoal and clay (fuller's earth) work well as adsorbents¹⁹⁻²².

Early treatment intervention to reduce absorption can be facilitated by early recognition of the signs of zinc phosphide toxicities (respiratory acidosis, metabolic acidosis, gastrointestinal irritation, abdominal discomfort, and chest tightness). As far as we are aware, this is the first incidence of zinc phosphide poisoning (also known as rodenticide) to be reported in the Indian state of Tamil Nadu. Despite advancements in therapy and supportive care, this case is reported to highlight the significant fatality risk associated with zinc phosphate poisoning²³⁻²⁶.

CONCLUSION:

The present patient was admitted to the hospital in critical condition. The patient's physical examination and routine tests, hemodialysis and gastric lavage were performed because of kidney damage, and the patient was closely monitored. As a result, patients who have stable hemodynamically are discharged.

The toxic effects of rat poisoning, a widely used rodenticide, are mediated through the conversion of phosphide to phosphine gas. Refractory hypotension and arrhythmias have been found to be the primary causes of most deaths, which happen in the first 12 to 24 hours. With repeated hemodialysis, the patient in the present case started to improve. The majority of peripheral hospitals' lack of supportive care and renal replacement treatments makes it extremely difficult to offer prompt interventions to stop mortality. Therefore, the chemical composition of the poison is important for the prognosis, as well as for close monitoring and quick action.

It is important for community health to educate the public about poisoning and the potential risks of zinc phosphate. The general public should be informed about poisoning and how to treat it at home. Because this cheap rodenticide is readily available and easy to obtain, it is important to take severe measures against it. This makes it a common poison used in purposeful self-harm or attempted suicide.

PATIENT INFORM CONSENT: While preparing a case report for publication patient's informed consent has been taken.

CONFLICT OF INTEREST: The Author declares that there are no conflicts of interest.

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