Case report: Poisoning is a common problem worldwide. Among the various poisonings, rat poison accounts for a significant number of cases. The common rat poisons are anticoagulant containing (superwarfarins) and phosphide containing. In case of superwarfin poisoning, the major symptom is increased prothrombin time and the management is directed towards coagulation. In case of phosphide containing, the symptoms are non-specific and the patient should be managed symptomatically. We present three case vignettes of rat poisoning cases treated at our hospital. Since rat poisoning is a common problem, the hospitals should also develop standard treatment guidelines regarding the management of rat poisoning. One should also make the differential diagnosis of the poisoning based on the type of rat poison prior to the management.

Keywords: Superwarfarin, prothrombin, Rodenticide

Consumption of rodenticides by humans

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Introduction

Rat poisoning is one of the common causes for poisoning in developing countries like Nepal. Data from developing countries suggest that rat poisoning is associated with significant mortality.1,2 Because of the lack of information regarding poison management, lack of availability of antidotes and other reasons the management of rat poisoning often poses a challenge to the clinicians. In this short review, the authors present three case vignettes of rat poisoning and provide an overview regarding the magnitude of the problem and its clinical management.

Case Vignette 1: A 14 year girl known case of seizure disorder on Carbamazapine was brought to Emergency Department with alleged history of consumption of Rodenticide one hour prior to admission and had two episodes of vomiting. Patient’s general condition was fair and vitals were stable. All her reports were normal except for prothrombin time raised by 4.18 seconds over control. She was managed conservatively and discharged on day three after psychological counseling.

Case Vignette 2: A 35 year male was brought to Emergency Department with complaints of vomiting, 15-20 episodes and abdominal pain and alleged history of consumption of rodenticide around twenty four hours prior to admission. Patient general condition was stable but his reports revealed increase of prothrombin time by 3 seconds over control and elevated hepatic enzymes. Rest of the investigations was normal. He was managed conservatively with supportive treatment. Patient improved clinically and was discharged on day six.

Case Vignette 3: A 22 year lady with no significant past history was brought to Emergency Department with alleged history of consumption of rat poison (time not known). Her vitals were stable and the routine investigations, including ECG was normal at the time of admission and was given supportive management. After 24 hours of admission patient developed severe pain abdomen with circulatory shock and sinus tachycardia. Clinical examination showed no signs of perforation. A diagnosis of Myocarditis was the strong suspicion and was given all the necessary inotropics and ventilator support but could not be revived and declared dead after 36 hours of admission.

Epidemiology of rat poison: Rat poisoning is a common problem. A study from India reported it to be the most common poisoning in a North Indian hospital accounting
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for 38.23% of the total poisoning cases. Another study from South India reported Rat poison to be responsible for 15% of the mortality due to poisoning. A study from Nepal identified rat poison to be the third most poisoning accounting for 6.55% of the total poisoning cases. In 2001, the American Association of Poison Control Centers reported more than 19,000 exposures to rodenticides. Most of these cases involved long-acting anticoagulants ingested by children less than 6 years of age.

**Types of rat poison:** Rat poisoning can be of several types; anticoagulant containing (superwarfarins), phosphide containing, vacer containing, strychine containing, thallium, barium, arsenic, cholecalciferol, phosphorous and red squid containing. The first two types are commonly reported and are hence discussed in this article.

**Signs and symptoms:** In case of superwarfarins, acute toxicity may be evidenced by transient abdominal pain, vomiting or heme positive stools. Some may have far longer action on vitamin K-dependent clotting factors than does warfarin. In case of phosphide containing rodenticides, the patients may present with nausea, vomiting, diarrhea, hypotension unresponsive to pressor agents and a rotten fishy or garlic odour.

**Mechanism of toxicity:** In case of superwarfarins, the compounds inhibit the hepatic synthesis of Vitamin K dependent clotting factors II, VII, IX and X. Only the synthesis of new clotting factor is inhibited and the effect of the ones already synthesized is not inhibited. Upon exposure to moisture, it liberates phosphine gas, which is absorbed rapidly by inhalation, dermally, or gastrointestinal. Phosphine gas is a highly toxic gas, especially to the organs of high oxygen flow and demand such as the brain, lungs, kidney, heart and liver. Phosphine is known to inhibit the electron transport chain in the mitochondria and causing the patho- physiological changes. The gaseous nature of phosphine has the potential for contamination of attending emergency service personnel too.

**Decontamination:** In case of superwarfarins, Ipecac syrup should not be administered. The efficacy of oral activated charcoal in preventing the absorption of superwarfarins has not been adequately studied. Transportation to an emergency department should not be delayed, in order to attempt activated charcoal administration. In case of phosphides too, activated charcoal should be administered if available. Ipecac induced vomiting may be beneficial if it can be given within a few minutes of exposure.

**Treatment:** The treatment of superwarfarin type rat poisoning include mainly the management of bleeding. If significant bleeding occurs, all the possibility of bleeding from any other part of the body should be considered. If there is an evidence of significant anticoagulation, Vitamin K1 should be given to the patient. Vitamin K1 treatment should be based on laboratory evidence of coagulopathy and not administered prophylactically. Administration prior to laboratory evaluation can delay the onset of anticoagulation and makes the diagnosis and laboratory monitoring more difficult. Vitamin K1 must be administered in high doses and at frequent intervals until clotting factor tests are normal (days or weeks), and the tests must be monitored for several months after they have initially returned to normal.

In case of poisoning due to phosphides, the management is mainly symptomatic. Severe hemolysis from phosphine gas (released from zinc phosphide) may require exchange transfusion of RBCs. Management of seizures and hypotension should be done if the patient develops these symptoms. However, the patients should be admitted and observed for at least 48-72 hours for delayed onset of pulmonary edema.

**Prognosis:** Anticoagulant rodenticide poisoning are usually relatively simple to treat and patients generally recover. Phosphide intoxications are potentially fatal. A five year study from North India reported 195 hospital admissions with aluminum phosphide ingestion of which, 115 died (58.97%).

**Psychological counseling:** Upon successful management, the patients should be counseled to prevent reattempts in the future.

**Conclusion**

Since rat poison is freely available and is associated with a significant mortality, this gains attention. The patients with rat poisoning should be treated immediately and should be provided an emergency and intensive care unit care as it is associated with a higher mortality. The hospitals should also develop standard treatment guidelines regarding the management of rat poisoning.

**References**


