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Clinical management of Zinc phosphide toxicity in German Shepherd dog

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Abstract

Zinc phosphide is an ingredient used in some rat poisons, and is also commonly used by pest control professionals. The study explores accidental ingestion of Zinc phosphide (Rodenticide) in three German Shepherd dog and its therapeutic management. All the three pups were treated intravenously with normal saline, dextrose, steroids, anticoagulant and multivitamins and orally drenched heamatinics and hepatoprotectant as follow up treatment according to the symptoms and severity of the toxicity. Out of three pups, two were recovered fully after post treatment, but one male pup could not recovered because of toxicity in multi-organs and died within 36 hours of post ingestion.

Keywords: Zinc phosphide, German Shepherd, hepatoprotectant, heamatinics

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Introduction

Zinc phosphide and aluminium phosphide are used extensively as a cheap and effective grain fumigant and rodenticide in developing countries (Christophers et al., 2002). Poisoning by these pesticide and rodenticide is one of the most common household dangers to our pet (Goel and Aggarwal, 2007). Zinc phosphide is an ingredient used in some rat poisons, and is also commonly used by pest control professionals. One of the effects of zinc phosphide on the body is the release of gases in the stomach, and the animal that has ingested zinc phosphide have garlic or rotten fish smell. Despite the extreme toxicity of phosphide, no effective antidotes are available, treatment is symptomatic (based on symptoms) and side effects of zinc phosphide poisoning can linger for several days after treatment.

In this case, we explored therapeutic management of accidental ingestion of zinc phosphide in German Shepherd dogs (GSD).

History and clinical sign

The Three GSD pups of four months old (1 male and 2 female) accidently ingested zinc phosphide of variable quantities. Clinical signs were observed in acute phase in one female and one male dog after 20 minutes of ingestion. The male dog had ingested large amount of rat kill than other two dogs, which showed more severe clinical signs including anorexia (loss of appetite), nausea, abdominal pain, colic, prostration, lethargyness, ataxia (loss of muscular coordination), chest tightness, dyspnea (frequent rapid breathing), salivation and garlic or fishy smell. The female dog had ingested little bit lower quantity showed less intense symptoms. Similar symptoms were also reported by various workers (Casteel and Bailey, 1985; Chugh, et al., 1998) in such type of pesticide and rodenticide toxicity. Second female apparently looked normal with slight drowsiness and salivation otherwise looked quite active.

On second day, the two female pups apparently looked normal and had normal feeding behaviour, but one male pup was still looking depressed, lethargic with similar symptoms as shown in first day and gradually the condition became worse from bad and could not able to cope up. On physical examination with abdominal palpation, the liver and spleen palpated were inflamed, and firmed in consistency.

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Treatment

The owner was advised immediately to drench common salt solution to all affected dogs to induce vomition. Of these two pups (1 male and 1 female) showed intense signs of poisoning. Immediately, intravenous infusion of normal saline (1 lit) and 5% dextrose (500 ml) solutions were injected to all three pups. Along with fluid therapy inj. Atropine sulphate at the rate of 4ml, inj. Dexomethasone at the rate of 3ml, inj. Vitamin K analogue (Kaplin) at the rate of 1ml I/V and 1ml I/M and inj. Multivitamin (Tribivet) at the dose rate of 2ml were given intravenously. During the intravenous fluid therapy, both the pups vomited and expelled out the entire ingested black coloured rat kill poison with stomach contents. The male dog has expelled large amount of black coloured vomitus as compared to female pups, suspected that male dog had ingested maximum amount of poisonous (Rat kill) material. On second day, one female became normal but other two were treated as above and were additionally given inj. Prednisolen at the dose rate of 1.5 ml I/M.

After completion of treatment, all the pups were drenched with liq. Cremafine at the rate of 20 ml to expel out the residual of the poisonous material through feces and haematinics Liq. Dexorange at the rate of 10 ml and liquid Sorbitol (sorbiline, Franco-Indian) for liver protection, regulation of hepato-digestive functions and metabolic disorder twice a day as follow up treatment for further 10 days. Both the female pups had been fully recovered after complete treatment, but the male pup could not be recovered, and died after 30 hrs of post-poison.

Discussion

Zinc phosphide is often recommended as the rodenticide of choice because it is fairly specific for rodents and there is no true secondary poisoning, except possibly in dogs and cats. Most animals that feed on rodents are unaffected because the zinc phosphide does not accumulate in the rodent's muscles or other tissues. Experimentally, several predators and scavengers have been exposed on a secondary nature, but only dogs and cats have been affected.

During this therapeutic treatment, two pups recovered successfully by treatment of intravenous administration of saline, dextrose, atropine, steroid and oral administration of heamatinics and hepatoprotectant but one male pup did not respond indicating multi organs toxicity (liver, spleen and kidney) and intravascular haemolysis. Gupta and Ahlawat (1995) and Aggarwal et al. (1999) reported metal phosphide effects on heart, lungs, kidneys and gastrointestinal tract because of cytotoxic phosphine gas production and intravascular haemolysis in patient in aluminium phosphide poisoning were reported, respectively. Zinc phosphide's toxicity is primarily due to liberation of phosphine gas (PH_3) (Rodenberg et al., 1989; Osweiler, 1996). The susceptibility to PH_3 occurs when food is ingested with the toxin because of concomitant gastric acid secretion in the stomach leading to more rapid phosphine gas release and enters into blood stream. This results in damage to the blood vessels and erythrocyte membranes and eventual cardiovascular collapse and irritation of the alimentary tract.

Conclusion

Metal phosphides poisoning is toxic to rodents and are used to protect the grains in stores and fields. Once administered in the body, a metal phosphide gets decomposed by dilute hydrochloric acid in stomach, and liberates highly toxic phosphine gas; the latter acts as respiratory and mitochondrial poison and ultimately lead to vital organs affection. Despite the extreme toxicity of metal phosphides no effective antidotes are available for treating victims of poisoning episodes.

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