Zinc Phosphide poisoning in Indian Jungle Myna *(Acridotheres fuscus)*

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Abstract

Zinc phosphide is used as a rodenticide and is toxic to animals with varying toxicity among species. Primary poisoning of wildlife has been recorded especially in seed-eating birds and waterfowl, secondary poisoning may occur if predators eat poisoned animals that contain a large dose in their alimentary canal. The episode of death of Jungle Myna (*Acridotheres fuscus)* was noted in the Nilgiri Biosphere of the Western Ghats by the locals and reported to the forest authorities. Subsequently four dead birds were collected for thorough post-mortem examination and samples were collected for laboratory investigation. The samples were subjected to various tests like hydro cyanic acid detection test, zinc phosphide detection test and organo-phosphorus compounds detection test. The history, the post-mortem findings and the toxicological investigation confirmed that these birds succumbed to the rodenticide, zinc phosphide. We confirm that the Jungle Myna opted for the zinc phosphide coated grains used for rodent control in the forest area.

Introduction

Zinc phosphide (Zn_3PO_2) is heavy, finely ground, crystalline dull greyish-black powder widely used as a rodenticide to control rats, mice, voles, ground squirrels, prairie dogs, nutria, muskrats and feral rabbits (Casteel S.W *et al*, 1986; Clarkson, 2001; Meister, 2001). Other species can be exposed to it accidentally or by intentional product misapplication. Zinc phosphide is toxic to birds, mammal species, and freshwater fish (Extoxnet, 1996; Knight, 2006). However, the toxicity varies among species (Albretsen, 2004). The lethal, oral dose of zinc phosphide for most domestic mammalian species is reported to be between 20-40 mg/kg body weight (Casteel S.W *et al*, 1986; Albretsen, 2004). The median lethal oral dose (LD_{50}) of zinc phosphide for rodents is reported to be 45.7 mg/kg (Clarkson, 2001). The minimum lethal oral dose for chickens when given with starch in gelatin capsules is 10 mg/kg body weight (Robertson *et al.,* 1945), whereas the oral LD_{50} for partridges and pheasants for zinc phosphide coated wheat is 26.7 mg/kg body weight (Janda and Bosseova, 1970). Among all avian species tested so far, white-fronted geese *(Anser albifrons)* are the most sensitive with an oral LD_{50} of 7.5 mg/kg for zinc phosphide coated hulled oats (Glahn and Lamper, 1983).

Primary poisoning of wildlife has been recorded, especially in seed-eating birds and waterfowl (Colvin, 1988). Secondary poisoning may occur if predators feed on poisoned animals that contain a large dose of zinc phosphide in their alimentary canal (Guale *et al.,* 1994). However risk of secondary poisoning is considered less because poison does not accumulate in body tissues for longer duration. There are several reports of wildlife and domestic animal intoxications following ingestion of zinc phosphide (Stowe *et al.,* 1978; Drolet *et al.,* 1996). Recently zinc phosphide intoxication in wild turkeys *(Meleagris gallopavo)* was reported by (Poppenga, *et al.,* 2005). However, there have not been any reports of intoxication in Jungle Myna *(Acridotheres fuscus).* It is primarily an insectivorous bird feeding on the ground and normally seek the company of cattle and horses, on which it readily perches. Aside from insects it feeds on fruit and soft vegetables.

Materials and Methods

In November 2008 four Jungle Myna carcasses were submitted by the animal husbandry/forest officials of Ooty range in Nilgiri Biosphere Reserve of the Western Ghats, with a history of sudden death. The symptoms of sudden death of limited number of birds, congested lungs and hemorrhagic liver did not suggest Avian Influenza and there were no death of other birds including backyard poultry in the particular area. Gross post-mortem examination revealed congested lungs, with hepatic and peritoneal hemorrhages. Feathers and green leaves were found in the gizzard, but no other significant lesions were observed. Stomach/gizzard contents (Fig-1), liver tissue (Fig-2) and loop of intestine were submitted for toxicological examination.

The samples were subjected for various tests like hydro cyanic acid test, zinc phosphide and organo-phosphorus compound (OPC) detection tests. For zinc phosphide detection the samples were processed following the method of Curry *et al., (*1958). About two grams of sample was ground using mortar and pestle after adding a small amount of distilled water. The ground mixture was transferred into a screw cap test tube and 2 ml concentrated hydrochloric acid was added to start the reaction. Then the tube was filled to three fourth with distilled water and the cap was plugged with a filter paper soaked in 1% silver nitrate solution and closed tightly so as to withhold released vapours, after placing the tube containing tissue suspensions in boiling water bath approximately for 10-12 minutes or until the sample in the tube starts boiling. Vapours from boiling tissue soak the filter paper plugged on to the tube cap, in the presence of phosphide, the silver nitrate soaked

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Fig. 1 Stomach/gizzard contents after post-mortem

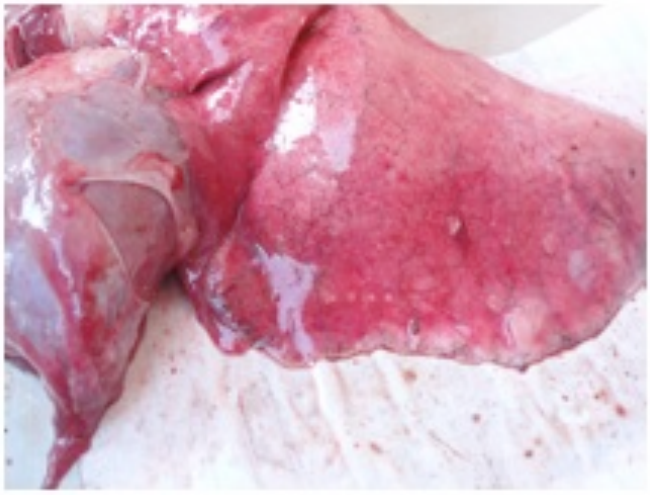

Fig. 2 Highly congested liver tissue

filter paper turns black. Filter paper was observed for interpretation.

For further confirmation the silver grey deposits of silver phosphide were collected from the filter paper after treatment with concentrated nitric acid. The dissolved material was transferred into a test tube and heated till boiling. Final interpretation of the substance was based on the development of canary yellow colour upon addition of ammonium molybdate solution.

Results

One of the widely followed approaches for diagnosis of zinc phosphide poisoning is the detection of phosphine gas, which is the reason for the fatality of zinc phosphide intoxication. The tests were interpreted based on the change in the colour of filter paper to silver grey, which was suggestive for the presence of zinc phosphide (Fig-3), and subsequently on the development of canary yellow precipitate in the confirmation test, which ascertained the presence of zinc phosphide (Fig-4). All other toxicological examinations were negative.

Fig. 3 Qualitative filter paper showing changes in the color of the filter paper to silver grey

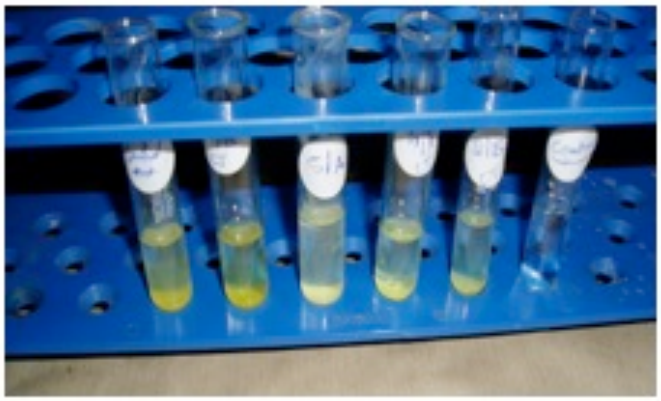

Fig. 4 Confirmatory test showing development of canary yellow precipitate

Discussion

Based on the history, post-mortem examination and toxicological investigation it was confirmed that these birds succumbed to the rodenticide zinc phosphide. There are several similar reports, which detected zinc phosphide by detection of phosphine gas (Guale *et al.,* 1994; Drolet *et al.,* 1996; Sterner, 1996). Sample preferred for confirmation are crop/gizzard/ stomach contents since other samples are unlikely to contain detectable concentrations (Robertson *et al.,* 1945; Matschke *et al.,* 1992; Guale *et al.,* 1994). Our study also confirms the suitability of the above indicated samples which is in accordance with the literature cited. Phosphine gas however dissipates rapidly in air and therefore, samples of gastrointestinal contents should be packed in air-tight containers and stored and shipped in a cold chain to laboratory as early as possible (Guale *et al.,* 1994).

Zinc phosphide releases phosphine especially in an acid environment (pH < 7). Hence the toxicity due to the release of phosphine is highest in the alimentary canal, particularly in the stomach, where the phosphine gets absorbed, causing oxidative tissue damage (Shivaprasad, 2001). Since phosphine gas reaches respiratory organs through haematogenous

route, damage to lung tissue is likely to be responsible for early deaths.

The onset of clinical signs following ingestion varies but occurs usually within four hours (Casteel *et al*, 1986). Partridges and pheasants developed clinical signs between two hours and six hours after ingestion, which indicates the acuteness of the toxicity (Janda and Bosseova, 1970). Clinical signs are nonspecific; experimentally poisoned poultry exhibited depression, ruffled feathers, anorexia, and diarrhea (Roberston *et al.,* 1945). The post-mortem lesions we found in our study were nonspecific and limited to generalized organ congestion and pericardial, pleural, and peritoneal effusions which is in line with findings of Robertson *et al.,* (1945).

Owing to the scarcity of food it was suspected that the Jungle Myna opted the Zinc phosphide coated grains etc which otherwise are used as rodenticide control measures by the tribal community living in the edges of the forest. Differential diagnosis of any wildlife mortality should always consider direct ingestion of Zinc Phosphide. Veterinarians and the general public should be aware of the dangers to wild birds as well as other animals when using rodenticides so that they can lend a helping hand in saving wild fauna.

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