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Therapeutic management of zinc phosphide poisoning in two non-descript bullocks

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Abstract
Two non-descript Bullocks from the Palodi village under Veterinary Dispensary Grade 1 Nimbhora from Akola District (Maharashtra) had accidental ingestion of zinc phosphide baits and developed signs of poisoning. Clinical examination of both the bullocks revealed bloat in one bullock and ataxia, struggling, weakness, dyspnea and elevated rectal temperature in both the bullocks. Pre-treatment hematological findings showed mild anemia in both the bullock. Each bullock was treated with 2 liters of 5% Sodium bicarbonate orally, Activated charcoal 1 gm/kg orally, Parenteral Ringers lactate and Calcium Borogluconate to overcome acidosis, Flunixin meglumine 1.1 mg/kg intramuscularly, Ofloxacin and Ornidazole bolus 2 BD orally, iron containing liver tonic 50 ml BD orally and probiotic bolus 2 BD orally. Both the bullocks showed clinical improvement from the evening on same day and recovered completely within eight days.

Keywords: Zinc phosphide, anemia, sodium bicarbonate, activated charcoal

Introduction
Zinc phosphide (Zn_3P_2) is commonly used inorganic compound in pesticide products as a rodenticide for the control of mice, rats and ground squirrels. Rodenticides containing zinc phosphide are used in both agricultural and residential settings. Formulations include bait pellets, granules, dust, and tracking powders [1]. Zinc phosphide is highly toxic to sheep, cows and goats as well as non-ruminants [2]. Rodent formulations contain 2% to 5% zinc phosphide mixed with grains; therefore, the baits are attractive to most species. The lethal dose of zinc phosphide in ruminants is approximately 20-50 mg/kg body weight [3].
The most common route of zinc phosphide poisoning or toxicosis in animals is by direct accidental ingestion of baits containing zinc phosphide. The present case report deals with clinical cases of zinc phosphide poisoning and its successful therapeutic management in two non-descript bullocks.

History, Clinical examination and Diagnosis
Two non-descript Bullocks from the Palodi village under Veterinary Dispensary Grade 1 Nimbhora from Akola District (Maharashtra) had accidental ingestion of zinc phosphide baits and developed signs of poisoning. Clinical examination of both the bullocks revealed bloat in one bullock and ataxia (Fig. 1), struggling, weakness, dyspnea and elevated rectal temperature in both the bullocks. Pre-treatment hematological findings showed mild anemia in both the bullock. Based on history, clinical examination and laboratory findings the cases were diagnosed for Zinc phosphoid poisoning.

Treatment and Result
In the present case, each bullocks was treated with 2 liters of 5% Sodium bicarbonate orally, activated charcoal 1 gm/kg orally, parenteral Ringers lactate and Calcium Borogluconate to overcome acidosis, Ofloxacin and Ornidazole bolus 2 BD orally and probiotic bolus 2 BD orally. As both the bullocks were anaemic, hence deworming with single dose of Albendazole 7.5 mg/kg and iron containing liver tonic at the dose of 50 ml BD orally were given for 15 days. Both the bullocks showed clinical improvement from the evening on same day and recovered completely within eight days.

Discussion and Conclusion
The toxicity of zinc phosphide is due to the production of phosphine gas by hydrolysis of phosphide to phosphine [4]. Zinc phosphide requires acidic conditions for appreciable hydrolysis

and subsequent formation of phosphine gas. In the present cases both the bullocks may be in the stage of sub-clinical ruminal acidosis (SARA) as farmer was regularly feeding them with rice, wheat and jowar flour. Sub-clinical ruminal acidosis in both the bullock promoted the production of Phosphine gas and subsequent signs of toxicity. Once released in the gastrointestinal tract, phosphine gas is absorbed along with zinc [5]. It Block the pathways of energy production and leads to cell death and eventual multiple-organ failure. Brain, heart, liver and kidney are most affected by phosphine gas due to high metabolic rates and high demands for oxygen.

Clinical signs occur in most species within 15 minutes to 4 hours of a toxic ingestion [6]. In cattle sign includes bloat, ataxia, weakness, prostration, dyspnoes, gasping, struggling, convulsions or hyperaesthesia, increase in body temp and coma leading to death which occurs in 3-48 hours [3]. In present case one bullock had bloat and elevated body temperature in both the bullocks. Subacute or chronic toxicities are not observed with posphides. Onset of signs may be delayed for up to 12 hours or more in animals who consumed the bait without any other food in their stomach.[7] Gastric acid release in animals that have recently eaten causes more rapid release of phosphine [8]. Phosphine gas rapidly enters the blood and is widely distributed to the lungs, liver, kidney and other organs.

Other possible mechanisms of action for phosphine include creation of hydroxyl radicals while simultaneously inhibiting catalase and peroxidase, corrosion of exposed tissues and metal toxicity from the zinc, magnesium or aluminum [9, 10]. Phosphine gas is readily released in acidic environments and can block cytochrome C oxidase, resulting in disruption of oxidative phosphorylation within mitochondria of cells [11]. Phosphine disrupts mitochondrial respiration once it is absorbed. Phosphine appears to block protein and enzyme synthesis [12]. More recent findings suggest that phosphine has an inhibitory effect on oxidative respiration and forms highly reactive free radicals [13]. The overall effect is a combination of local corrosive effects in the gastrointestinal tract and circulatory collapse. Death occurs from pulmonary edema or cardiac arrest.

Diagnosis of zinc phosphide poisoning is based on history of exposure to zinc phosphoid and suggestive clinical signs. Analysis of phosphine in tissues is not readily available but its presence in stomach content may be detected by its acetylene odor, Haematological and biochemical alteration are inconsistent [14].

There is no specific antidote for zinc phosphide poisoning. Therefore the treatment is aimed by administration of symptomatic and supportive drugs. Supportive therapy should be aimed at correcting acidosis, treating shock and liver failure, and controlling seizures. In asymptomatic cases, gastric lavage followed by activated charcoal or a di-tri-octahedral smectite has been used successfully [13]. In the present case Sodium bicarbonate 2 litres of 5% orally to neutralize acidity, activated charcoal 1 gm/kg orally, and calcium borogluconate and RL to overcome acidosis [3]. With the Ofloxacin and Ornidazole bolus orally, probiotic bolus and iron containing liver tonic orally were given. Both the bullocks showed good recovery from same day and recovered completely within eight days.

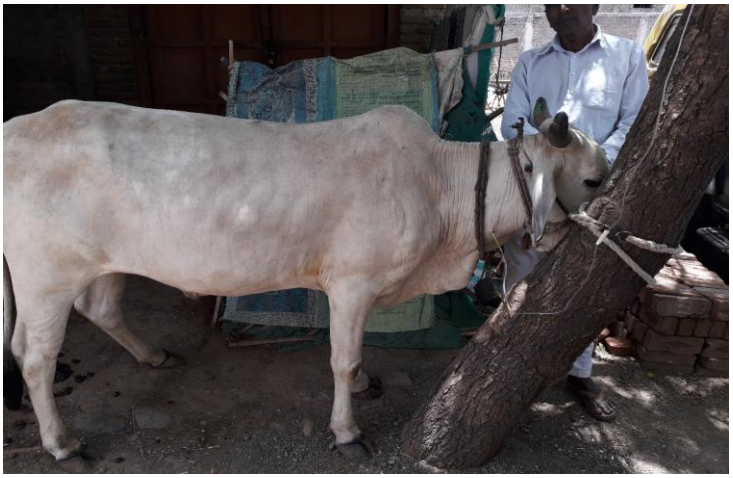

Fig 1: Bullock standing with his limbs apart from each other (Treatment at Farmers Home)

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