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Sorghum poisoning in buffaloes and its treatment

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Abstract
Sorghum (Jowar/Chari) is an important feedstuff for livestock. A negative characteristic of sorghum from an animal health perspective is its capabilities to cause cyanide poisoning. Cyanide can cause significant animal health problems including death. In the present study deals with an outbreak of sorghum poisoning from Nirdhari village of the Mandsaur district of the Madhya Pradesh in the month of May 2018 with a history of accidental ingestion of sorghum crops. A total of eight buffaloes were exposed, three animals found dead and five were severally affected with clinical symptoms like rapid and difficult breathing, dry noses, dilated pupils, motionless eyes, congested mucous membranes, muscle tremors, convulsions and recumbency. On post-mortem examination revealed that carcasses were bright cherry-red coloured blood, congestion of liver, lungs, kidney and congestion and petechial haemorrhages on the surface of the gastrointestinal tract. Based on history and clinical symptoms, affected animals were treated with sodium thiosulfate @ 25 gm/100 kg body weight intravenously. After medication animals were recovered within a day.

Keywords: Sorghum poisoning, buffaloes, clinical signs, post-mortem lesions and treatment

Introduction
Cyanogenic plant toxicity is one of the most common plants poisoning among the grazing livestock [1]. Most common source of cyanogenic plant poisoning in India is feeding of immature sorghum *i.e.* young shoots of *Sorghum vulgare* and *Sorghum sudanense* to livestock [2]. Cyanide does not occur freely in cyanogenic plans. Instead, they contain cyanogenic glycosides whose molecules contain a cyano group (--CN). It is cyano group that is a source of cyanide. Cyanogenic plants contain cyanoglycosides and enzymes within the cells that can remove the cyano group from cyanoglycosides. When the plant is frozen, chopped or chewed, damaged plant cells can release enzymes in their vacuoles, this enzymes and cyanoglycosides may contact with each other and produce cyanide [3]. Ruminants are more susceptible than monogastric animals because ruminal microorganisms can rapidly break plant cells and release cyanide [4]. Once cyanide is release from cyanoglycosides, it can be absorbed quickly into the bloodstream and circulated, then it's merged with methemoglobin and forms cyanomethemoglobin. In the bloodstream, cyanide inactivates cytochrome oxidase enzyme by binding ferric (Fe^{+++}) iron which is within this enzyme [5]. Because of that, the enzyme cannot combine with oxygen and electron transportation becomes inhibited. The patient cannot use caloric oxygen and cellular respiration stops immediately [6, 7]. Hydrogen cyanide (HCN) poisoning is related to the amount of forage consumed and the animal's physiological condition, but HCN levels exceeding 220 ppm on a wet weight (as is) basis is dangerous. Forage containing <100 ppm HCN, wet weight, is usually safe to pasture [8]. On a dry weight basis analysis, forages with more than 500 ppm HCN should be considered potentially toxic [9]. Cyanide is very toxic to mammals and its effects may be noticed within 5-15 minutes after consumes the cyanogenic plant. The brain and heart are the first to be affected by lack of oxygen and so the resulting clinical signs prior to death include excessive salivation, difficulty breathing, mucous membranes are bright red in colour, muscle tremors, convulsions, collapse and death from respiratory paralysis [1, 5, 10]. The present study is designed for the investigation of sorghum poisoning in buffaloes and its treatment.

Materials and methods
Mandsaur is the city in the Malwa region and north-west district of Madhya Pradesh state of central India is famous for lord Pashupatinath temple and agriculture especially foropium cultivation. Generally, the climate of the Mandsaur is dry except in south-west monsoon. Monsoon season starts from mid-June to September. The cold season is from December to February. This is followed by the hot season from March to the middle of June. Farmers of this district are specially grown sorghum crop for animal feeding in the summer month. Hydrogen

cyanide (HCN) / Prussic acid poisoning can occur when livestock are pastured on sorghum-type plants, including grain sorghum, forage sorghum, sudangrass, sorghum-sudangrass crosses, Johnson grass, sweet sorghums, and other sorghum-type plants [2, 4, 5]. HCN poisoning is one of the most toxic and rapidly acting poisons [8]. In the present investigation, a total of 8 buffaloes were exposed to cyanogenic plant poisoning with a history of accidental ingestion of sorghum in the Nirdhari village of the Mandsaur district of the Madhya Pradesh on 17 May 2018. According to the owner, in morning time animals were uncontrolled graze of immature jowar fodder on his field. We were reached within hour to victim place and examined, 3 buffaloes (one male calf, one male and female adult buffalo) were dead and 5 buffaloes (adult female) severally affected with clinical symptoms like rapid and difficult breathing, dry muzzle, salivation, dilated pupils, motionless eyes, congested mucous membranes, bloat, muscle tremors, convulsions and recumbency. Based on history and clinical symptoms it was suspected as HCN poisoning case and affected animals were treated with sodium thiosulfate intravenously. On post-mortem examination revealed that carcasses were in the reasonable flesh, showing no external lesions. After the opening of carcasses, bright cherry-red coloured blood was observed. Rumen was bloated and filled with fresh green jowar material, congestion and petechial haemorrhages were observed on the gastrointestinal tract. Haemorrhages were observed on surface of the heart, muscles, congested and enlarged liver. Muscles colour was changed whereas no changes occur in the spleen. All the clinical signs, post mortem lesions and response to treatment shown by affected animals were typical of cyanide poisoning.

Results and Discussion

On the basis of history, clinical symptoms, postmortem lesion and response to treatment, it was diagnosed as a case of HCN poisoning. Similar types of investigation were reported in cattle by Gurnsey *et al.* [6], Nobrega *et al.* [1], Patel *et al.* [10]. Various animal species react differently when fed cyanogenic plants containing glycosides. These differences are caused by different anatomical structures and different detoxifying abilities of various animals. Cattle and buffaloes, both being ruminants, are known to be subject to poisoning by cyanogenetic glycosides. In the ruminants, pH of the rumen is neither strongly acid nor alkaline, and it contains a large flora of micro-organisms and considerable quantities of enzymes. The Rumen is an excellent medium for the hydrolysis of the glycosides with the liberation of the toxic agent HCN which is then rapidly absorbed into the blood. Non-ruminants have only one stomach which is strongly acid due to the presence of hydrochloric acid [11]. The hydrochloric acid reacts with the liberated HCN to form much less toxic substances formic acid and ammonium chloride. In the present investigation clinical symptoms of sorghum poisoned animals shown an increased rate of respiration, gasping, dry muzzle, nasal discharge, dilated pupils, motionless eyes, slightly bloated, slow heart rate, congested mucous membranes, anxiety and restlessness followed by depression, muscle tremors, convulsions and recumbency. These findings are in agreement with Lorgue *et al.* [12], Sargison *et al.* [13], Patel *et al.* [10] who reported in cattle. Clinical symptoms of HCN poisoning usually occur 15–20 minutes after the toxin is consumed. Death occurs very quickly, approximately 2–3 minutes after the onset of clinical signs in per acute cases, and within 1–2 hours in acute cases. On post-mortem examination revealed that carcasses were in the reasonable flesh, showing no external lesions. After the opening of carcasses, bright cherry-red coloured blood was observed. Rumen was bloated and filled with fresh green jowar material, congestion and petechial haemorrhages were observed on the gastrointestinal tract. Haemorrhages were observed on surface of the heart, muscles, congested and enlarged liver. Muscles colour was changed whereas no changes occur in the spleen. These type of lesions were also reported in cattle by Nobrega *et al.* [1], Nicholson, [5]. The clinical symptoms of cyanide poisoning are similar to nitrate toxicity, but animals with cyanide poisoning have bright red blood that clots slowly, whereas animals poisoned with nitrate have dark, chocolate-colored blood, blue mucous membranes, frequent urination and dilated and bloodshot eyes. Animals treated with methylene blue may recover. Stressed plants (cultivated, frosted, wilted) are most dangerous for nitrate. Regrowth of grain sorghum crops is probably the most dangerous for cyanide [14, 15]. In the present investigation severally affected animals were treated with sodium thiosulfate (@ 25 gm/100 kg body weight) mixed with 500 ml dextrose normal saline and given intravenously. After treatment of animals were recovered within a day. Similar treatment was given in sheep by Burrows and way, [16]. However, the antidote of choice in most of animals is combination of sodium nitrite and sodium thiosulfate [17, 18]. Sodium nitrate releases the cyanide from the cell (promotes the formation of methamoglobin), which binds with the sodium thiosulfate to form a non-toxic complex (forms thiocyanide) that is excreted. Non-specific supportive treatment, including respiratory stimulants and artificial respiration are unlikely to have any effect on the course of the disease. Many times treatment comes too late and the animal cannot be saved. Total 150-200 g sodium thiosulfate is also advocated orally in cattle. Oral doses of 30 g of sodium thiosulfate repeated at hourly intervals are also given orally to cattle [18].

Conclusion

The present investigation revealed that the sorghum poisoning in buffaloes is fatal. Keeping in view these factors, strategic treatment and control programme may be formulated to control plant poisoning in buffaloes in Mandsaur district and elsewhere in Madhya Pradesh.

References

1. Nóbrega Jr J, Riet-Correa F, Medeiros RM, Dantas AF. Poisoning by *Sorghum halepense* (Poaceae) in cattle in the Brazilian semiarid. Pesquisa Veterinária Brasileira. 2006; 26(4):201-204.
2. Vadlamudi VP. Cyanogenetic Plants. In: Veterinary Toxicology (Ed. S. K. Garg), 1st edition, CBS Publishers, New Delhi, 2000, 87- 91.
3. Harborne JB. Cyanogenic Glycosides and Their Function. In: Phytochemical Ecology. Academic Press, London, 1972, 104-123.
4. Knight AP, Walter RG. A guide to plant poisoning of animals in North America. International Veterinary Information Service (www.ivis.org), Ithaca, New York, USA, 2002, 5.
5. Nicholson SS. Cyanogenic plants. In: Veterinary Toxicology: Basic and Clinical Principles (Ed. Gupta, R. C.). Academic Press, USA, 2007, 873- 875.
6. Gurnsey MP, Jones WT, Merrall M, Reid CS. Cyanide poisoning in cattle: two unusual cases. New Zealand Veterinary Journal. 1977; 25(5):128-30.

7. Kutay Y, Banu D, Remzi G, Erman MO. Cyanide Poisoning in Cattle. Dairy and Veterinary Science Journal. 2017; 1(4):555-567.
8. Schneider NR. Overview of Cyanide poisoning. The Merck‟ s Veterinary Manual Online. (Available online http://www. merckmanuals. com/ vet/toxicology/cyanide poisoning/ overview_of_ cyanide_ poisoning. html, last updated March, 2012.
9. Fjell DL, Blasi DA, Towne G. Nitrate and prussic acid toxicity in forage: causes, prevention, and feeding management. Departments of Agronomy & Animal Science [Kansas State University], 1991.
10. Patel HB, Mody SK, Modi CM, Patel HB, Parekar SS. Treatment of Cyanide Poisoning in Crossbred Cows: A Profile of Drug Synergism. International Research Journal of Pharmacy. 2012; 3(10):169-170.
11. ATSDR (Agency for Toxic Substances and Disease Registry). Toxicological Profile for Cyanide. U.S. Department of Health and Human Services, Atlanta, US, 2006, 1-131.
12. Lorgue G, Lechenet J, Riviere A. In: Clinical Veterinary Toxicology. Blackwell Science Ltd. UK, 1996, 136-137.
13. Sargison ND, Williamson DS, Duncan JR, McCance RW. *Prunus padus* (bird cherry) poisoning in cattle. Veterinary Record, 1996, 138(8).
14. Dodd DC, Coup MR. Poisoning of cattle by certain nitrate-containing plants. New Zealand Veterinary Journal. 1957; 5(2):51-4.
15. Carrigan MJ, Gardner IA. Nitrate poisoning in cattle fed sudax (Sorghum sp. hybrid) hay. Australian Veterinary Journal. 1982; 59(5):155-7.
16. Burrows GE, Way JL. Cyanide intoxication in sheep: therapeutic value of oxygen or cobalt. American Journal of Veterinary Research. 1977; 38(2):223-7.
17. Clarke ML, Harvey DG, Humphreys DJ. Veterinary Toxicology, 2nd edition (ELBS) Bailliere Tindall, London, 1981, 175-178.
18. Radostits OM, Gay CC, Hinchcliff KW, Constable PD. Veterinary Medicine, A textbook of the disease of cattle, sheep, goat, pigs and horses, 10th ed. Saunders Elsevier London, New York, 2007, 1852-55.