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Research Article

TREATMENT OF CYANIDE POISONING IN CROSSBRED COWS: A PROFILE OF DRUG SYNERGISM

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ABSTRACT

Five cross breed cows suffering from cyanide poisoning were treated. Immaturely cut sorghum grass in pre monsoon season fed to animals was identified as a cause of poisoning. All the animals exhibited the severe clinical signs of poisoning like dyspnoea, anxiety, restlessness, staggering gait as well as tremors. All five cows were treated immediately at nearby farms where they were kept. While treating cows, three among five were administered saturated jaggery liquid prior to literature based allopathic SID treatment of 15 gm IV administration of Sodium nitrite and Sodium thiosulphate combination, where as rest two were given only allopathic treatment of Sodium nitrate and Sodium thiosulphate combination (15 gm IV). Cows which were treated with both jaggery and allopathic therapeutics recovered fast as compared to cows treated with allopathic therapeutics alone. Result suggests that jaggery play a synergistic action with allopathic therapeutics and responsible for early recovery from clinical symptoms of poisoning.

Key word: Cyanide, Crossbred Cows, Toxicity

INTRODUCTION

The plants, the most important source of cyanide toxicity in animals, which contain hydrocyanic acid (HCN) either free or in form of cyanogenic glycosides are called as cyanogenetic plants[1]. The glycoside itself is non-toxic, which owe its toxicity to cyanide after hydrolysis. Cyanogenetic plant toxicity is one of the most common plant poisoning among the grazing livestock. Ruminants are more susceptible to poisoning by cyanogenic plants, which release hydrogen cyanide than horses and pigs due to more efficient hydrolysis of the cyanogenic glycosides[2]. Within the group of ruminants, goats appear to be the most susceptible to cyanide. Based on the available data the following intake mg hydrogen cyanide (HCN) equivalents/kg b.w. per day) seems to be tolerated by the following animal species: pigs (2.9 mg/kg per day), poultry (2.8 mg/kg per day), ruminants (on the basis of goat studies) (0.25 mg/kg per day) and horses (0.4 mg/kg per day), respectively[3]. Cattle and buffaloes are also highly susceptible species. This type of toxicity usually occurs in animals when animal ingests large amount of immature sorghum fodder at pre-bloomed stage[4]. Factors that incur likelihood of HCN poisoning from ingestion of cyanogenic plants include: (1) large amount of free HCN and cyanogenic glycoside in plant, (2) rapid ingestion; (3) ingestion of a large amount of plant, and (4) ruminal pH and microflora that continue to hydrolyze glycosides to release hydrogen cyanide. Rapid intake of plant equivalent to about 4 mg HCN/kg of body weight is considered to be lethal amount of plant material[5]. In current trend for earning more profit, farmers are trying to cultivate more crops throughout year, so they may cut the forrage in immature stage, which is the main source of cyanide toxicity. Generally it happens due to lack of forrage for feeding to animals because of improper forrage management and also sometimes it may occur due to accidental ingestion while grazing[6].

About 120 plant species containing cyanogenetic glycosides are found in India. The plant material containing over 20 mg HCN per 100 gm is potentially toxic to livestock[1] (Garg, 2004). The details about toxicity index of HCN are given in Table 1.

The most common source of cyanogenic plant poisoning in livestock is feeding of green Sorghum fodder, particularly immature plant or young shoots. Details of plant forrage prone to cause HCN poisoning in animals is shown in Table 2.

Table 1: Level of HCN in forage (dry matter basis) and potential effect on animals[7]

Sr. No.	HCN/kg feed (ppm)	Effect of HCN on animals
1.	0-500	Generally safe
2.	600-1000	Potentially toxic
3.	>1000	Dangerous to cattle and usually cause death

Table 2: Millet and sorghum types and their potential cyanide accumulations[7]

Sr. No.	Type of plants (Millet or Sorghum)	Potential of Cyanide Toxicity
1.	Grain sorghums	High to very high
2.	Johnsongrass	High to very high
3.	Shattercane	High
4.	Forage sorghums	Intermediate to high
5.	Sorghum-sudangrass hybrids	Intermediate to high
6.	Sudangrass hybrids	Intermediate
7.	Sudangrass varieties	Low to intermediate
8.	Pearl and Foxtail millet	Very low

Present case report reflects incidence of cyanide toxicity in crossbred cows.

MATERIALS AND METHODS

Recently, one incidence of cyanide poisoning was observed in five crossbred cows in Sabarkantha district (Gujarat). After detailed history obtained from animal owner, it was revealed that the immaturely cut sorghum grass in pre-monsoon season fed to animals was the cause of poisoning.

All the animals in present study have shown the severe clinical signs of poisoning like dyspnoea, anxiety, restlessness, tympany, signs of colic, excitement, hypersalivation, bright red colored mucus membranes, dilated pupils, staggering gait as well as tremors. All the signs shown by the animals were typical to the cyanide poisoning. Similar type of signs were observed in cattle after *Sorghum halepense* poisoning in Brazil[8] and after experimental poisoning of *Amelanchier alnifolia* in Canada[9] whereas Tegzes *et al.* (2003)[10] also reported similar symptoms in goats after ingestion of *Hetromeles arbutifolia*.

RESULT AND DISCUSSION

Incidences of Cyanide intoxication were reported in beef cattle caused by the ingestion of *Glyceria grandis* S. Wats. *ex* Gray (Tall Mannagrass), in British Columbia[11] and in goats in California after eating clippings from California Holly *(Heteromeles arbutifolia)* which contains cyanogenic glycosides. Within four hours of feeding, three animals died, and seven were moribund[10].

Cyanogenic glycosides are hydrolyzed by β-glycosidases with the release of hydrogen cyanide. The glycosides are stored in vacuoles within plant tissues, whereas the hydrolytic enzymes are found in the cytosol. Damage to the plant from mastication, frost, drought, or trampling results in the combination of the enzymes with the glycosides, causing the formation of hydrogen cyanide. Additionally, rumen microorganisms produce the hydrolysis enzymes. Ruminants are therefore more susceptible to the toxic effects of cyanide than monogastric animals[10].

Hydrogen cyanide is absorbed readily from the gastrointestinal tract and enters individual cells. It acts at the cellular level by inhibiting mitochondrial oxidation–reduction reactions, disrupting cellular respiration. Specifically, it binds to cytochrome oxidase, the terminal step in electron transport necessary for cellular respiration and adenosine triphosphate production. Adenosine triphosphate formation is impaired, and tissues become depleted of energy. The effects are most noticeable in cells with a high metabolic rate and demand for oxygen, namely neurons and cardiac myocytes. Thus, the earliest signs after cyanide exposure are due to dysfunction in neurons and in the myocardium. This is manifested most commonly as collapse. Hypotension and tachycardia may be observed. Further damage to neurons arises secondary to systemic hypoxia and ischemia. Dyspnea also may be observed as the respiratory musculature becomes affected[12].

The treatment of cyanogenic plant poisoning is specific and must be initiated immediately after diagnosis. All five cows were treated immediately at nearby farms where they were kept. While treating cows, three amongst five were administered saturated jaggery liquid prior to literature based allopathic SID treatment of IV administration 3 gm of sodium nitrite and 15 gm sodium thiosulphate combination in 20 ml of distilled water. The other two cows were treated only with allopathic treatment of 3 gm sodium nitrite and 15 gm sodium thiosulphate combination in 20 ml of distilled water by IV route[4,6].

The result of the treatment shows that the cows, which were treated with both jaggery and allopathic therapeutics, were recovered fast as compared to cows treated with allopathic therapeutics alone. From the result it is concluded that jaggery plays a synergistic action with allopathic therapeutics and may enhance faster recovery from clinical symptoms of cyanide poisoning.

Following precautions are suggested to farmers to reduce the incidences of cyanide poisoning in animals as per guideline of Hawkins (2007)[13].

1. Animals should be fed with flowering stage or matured sorghum.
2. Prevent the access of animals to immature / wilted / frost bitten plants as it contains more amount of cyanogenic glycosides.
3. Care is to be taken if urea or other nitrogen fertilizers has been recently used, or if phosphorus levels are low.
4. Animals should be provided with a sulphur lick (10% sulphur in a salt lick) as sulphur plays an important role in detoxification of cyanide released in the digestive process.
5. Animals are to be supervised for the first 48 hours when introducing stock on to sorghum.

CONCLUSION

The use of knowledge of folklore medicine in treatising trivial as well as life threatening diseases of domestic animals is rational, economic as well as far safe from side effects and residue problem of synthetic medicines. But all folklore medicines must be evaluated for its synergistic action.

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