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Sorghum Poisoning in Cattle and its Therapeutic Management

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ABSTRACT

Sorghum is a cyanogenic plant and when its level exceeds 20 mg per 100 g (200 ppm, 200 mg/kg) of forage, liver enzymes gets exhausted and HCN starts accumulating in the animal body. When the cyanide level becomes 0.5-3 mg/Kg b.wt., clinical symptom of poisoning appears which may end up in death of the animal. The article reports about a cow that have accidentally poisoned with sorghum forage, the animal was treated with sodium nitrite, which is effective in converting haemoglobin to methaemoglobin, which in turn reacts with cyanide (Prussic acid) to form cyanomethaemoglobin. A simultaneous injection of sodium thiosulphate provides sulphur to convert cyanomethaemoglobin to non-toxic thiocyanate which is excreted through urine. This paper presents the successful treatment of cyanide toxicity in cattle.

KEYWORD

Asphyxiation; cyanogenic; glycosides; hydrocyanic acid; forage

INTRODUCTION

The most important source of cyanide toxicity in animals are plants which contain cyanogenic glycosides and these are called as cyanogenic plants. These glycosides are converted to Hydrogen cyanide (HCN) by hydrolisation in the body and responsible for toxicity. Cyanogenic plant toxicity is one of the most common plants poisoning among the grazing livestock. Ruminants are more susceptible to poisoning by cyanogenic plants due to more efficient hydrolysis of the cyanogenic glycosides to HCN than other animal and their feeding style. Cattle and buffaloes are highly susceptible but among ruminants, goats appear to be the most susceptible to cyanide.

About 120 plant species containing cyanogenic glycosides are found in India. Among these sorghum, Sudan grass, Johnson grass etc. are the plants that contain high amounts of glycosides which results in severe poisoning (Vogel *et al.*, 1987).

This type of toxicity usually occurs in animals when animal ingest large amount of immature sorghum fodder at pre-bloomed stage. Immature, wilted and draught affected plants are highly cyanogenic than the mature and normal plants. Rapid intake of plant equivalent to about 4mg HCN/Kg of body weight is considered to be lethal amount of plant material. In recent economic trend for earning more profit, farmers are trying to cultivate more crops throughout year, so they may cut the forage in immature stage, which is the main source of cyanide toxicity. Generally, it happens due to lack of knowledge of farmer and lack of forage for feeding to animals because of improper forage management and also sometimes it may occur due to accidental ingestion of immature, wilted and draught affected plants. The plant material containing over 20mg HCN per 100gm is potentially toxic to livestock (Garg, 2004). The cyanogens are hydrolysed by rumen microorganism resulting in release of highly toxic substance Hydrocyanic acid (HCN) or prussic acid. HCN releases cyanine which inhibits cytochrome oxidase enzyme thereby preventing oxygen supply into the cell, so death of animal due to severe asphyxia. Thus, Present case study deals with successful therapeutic management of sorghum poisoning in cattle.

MATERIALS AND METHODS

During the month of May 2017, a farmer from Chandmari village of Patna district (Bihar) reported that his two cows aged between 5 to 6 years were left for grazing nearby plot having immature, wilted and draught affected sorghum forage. Owner observed a sudden death of one cow near sorghum field with accelerated and deep respiration and the other cow suffered with severe illness. He revealed that his cows were healthy before allowing for grazing and he was shocked for losing one cow suddenly and seeing other one was severely ill. On clinical examination affected animals revealed tympany, bright red coloured mucus membrane, dilated pupil, rapid breathing with mouth kept open, rapid weak irregular pulse, increased salivation and lacrimation, involuntary urination and defecation with muscular tremors. All the signs were typical to the cyanide poisoning. Similar type of signs was observed in cattle after *Sorghum halepense* poisoning in Brazil and after experimental poisoning of *Amelanchier alnifolia* in Canada whereas Tegzes *et al.* (2003) also reported similar symptoms in goats after ingestion of *Hetromeles arbutifolia.*

RESULTS AND DISCUSSION

Based on the history of feeding, field inspection, nature and severity of clinical

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symptoms, it was diagnosed to be a case of sorghum poisoning (Hydrocyanic acid toxicity). The sick cow was treated immediately. The treatment was initiated with injection of sodium thiosulphate (3 %) @ 660 mg/Kg b.wt. IV along with intra ruminal administration of 40 g to detoxify the remaining HCN in the rumen. Simultaneously injected sodium nitrite 1% solution @ 22 mg/Kg b.wt by IV route (Radostits *et al.*, 2000). Two litres of 5% Dextrose and 2 litre of Ringer's lactate were administered IV to dilute the toxin and as a supportive therapy. Injection Anistamin[a] (Chlorpheniramine maleate 10 mg/ml) @ 10 ml IM and Curadex[b] (Dexamethasone Sodium 4 mg/ml) @ 10 ml IM was given to take care of allergic reaction and to prevent from shock. After 3 hr of treatment the symptoms gradually disappeared and the cow showed interest towards the feed and water. Owner was advised to provide fresh ragi (Finger millet) straws, which will help in diluting the amount of poison in the rumen. The supportive treatment was continued for the next two days.

Sodium nitrite will convert haemoglobin to methaemoglobin, which react with cyanide (prussic acid) to form cyanomethaemoglobin. A simultaneous injection of sodium thiosulphate provides sulphur to convert cyanomethaemoglobin to the non- toxic thiocyanate, which is excreted in urine.

Normally cyanogenic glycosides of plants are converted to Hydrogen cyanide (HCN) by hydrolisation in the animal body and get metabolized by liver enzyme and converted into thiocyanate, which gets excreted in the urine. When cyanogens level in the plant exceeds 20 mg per 100 g (200 ppm, 200 mg/kg) of forage, liver enzymes exhausted and HCN starts accumulating in the animal body and those plants are potentially toxic to all animals (Gupta, 2012). When the cyanide level becomes 0.5-3 mg/kg body weight (depends on the animal), toxic symptoms starts appearing which may end up in death of the animal. The present paper represent the recovery of cattle is in co-ordination with reports by Burrows and Way (1979) and Buck *et al.* (1988).

a - Brand of Intas Animal Health, Ahmedabad, b- Brand of Concept Animal Health, Mumbai

CONCLUSION

Cyanide poisoning is one of the most important poisonings in ruminants and its effects may be noticed within 15 to 20 minutes to as long as a few hours depending on the concentration of cyanogenic glycosides and amount of cyanogenic plants consumed. Especially in cattle's pica ability, it became more dangerous. Treatment must begin rapidly because death can occur within few minutes during severe toxicity. So prevention is better than cure. To reduce the incidence of cyanide poisoning, farmers are suggested to prevent the access of immature/wilted/frost bitten plants as it contains more amount of cyanogenic glycosides. Only mature sorghum at flowering stage may be suggested for feeding of animals.

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