INTERNATIONAL RESEARCH JOURNAL OF PHARMACY
www.irjponline.com
ISSN 2230 – 8407

Research Article

HEVEA BRASILIENSIS POISONING IN MALNAD GIDDA CATTLE, KARNATAKA, INDIA

Lohith T.S*, Venkatesha M.D, Mallinath K.C, Sobharani M, Shankar B.P.
Southern Regional Disease Diagnostic Laboratory, Institute of Animal Health and Veterinary Biologicals, Hebbal, Bangalore, India
*Corresponding Author Email: lohithvetpharma@gmail.com

Article Received on: 10/06/14 Revised on: 30/06/14 Approved for publication: 09/07/14

DOI: 10.7897/2230-8407.0507117

ABSTRACT
Hevea brasiliensis, the rubber tree, is a tropical tree belonging to the family Euphorbiaceae, native to Amazon basin in Brazil and most economically important member of the genus Hevea, because of the milky latex extracted from the tree is the primary source of natural rubber. The young leaves of this tree are poisonous to most animals. In India it is found in areas where plantation is done for the commercial purpose. Hence poisoning in ruminants due to rubber tree is a rare phenomenon unless they have access to it. In the present investigation there were deaths of six adult Malnad Gidda cattle in Hale Ikkeri village of Shimoga, Karnataka, India in a span of two days. An attempt has been made to know the cause of sudden mortality in Malnad Gidda Cattle. Post-mortem examination was conducted on carcasses and samples were collected for the detection of toxic principle, Hydrocyanic acid (HCN) by Picric acid paper spot test. The young rubber tree leaves showed positive for the presence of toxic principle HCN. HCN is one of the most potent, rapid-acting poisons known. When a lethal dose is consumed, animals die from asphyxiation in a few minutes. Deaths in animals due to HCN poisoning, can be reduced by administering with a mixture of sodium thiosulfate and methylene blue.
Keywords: *Hevea brasiliensis,* Rubber tree, Malnad Gidda cattle, HCN, Asphyxiation

INTRODUCTION

Cyanides are found in plants, fumigants, soil sterilizers, pesticides/rodenticides and in a variety of cyanide salts used in industrial processes. Toxicity can result from accidental, improper, or malicious use or exposure, but in the case of large animals or livestock, the most frequent cause of poisoning is ingestion of plants that contain cyanogenic glycosides. The cyanogenic glycosides in plants yield free hydrocyanic acid, otherwise known as prussic acid. Hydrocyanic acid (HCN), normally is not present in plants. However, several common plants can accumulate large quantities of cyanogenetic glycoside. When plant cells are damaged by wilting, frosting or stunting, the glycoside degrades to form free HCN. Conditions in the rumen also favour degradation of the glycoside to free HCN[1]. Thus plants that contain the glycoside have the potential to cause HCN toxicity when consumed by ruminants. In India, plants most likely to cause HCN poisoning are sorghums whereas *Hevea brasiliensis* poisoning is rare phenomenon unless animals have access to it. In the present study an attempt has been made to know the cause of sudden mortality in Malnad Gidda Cattle.

MATERIALS AND METHODS

During the period of investigation it was informed that the animals had accessibility to young rubber tree leaves. The affected animals were examined for the presence of any abnormal clinical signs. Post-mortem examination was conducted on carcasses as per standard protocol. Post-mortem samples of liver, kidney, ruminal contents and the young rubber tree leaves (Figure 1) were collected in ice box for the detection of toxic principle, Hydrocyanic acid (HCN). Presence of hydrocyanic acid was done by Picric acid paper spot test[2]. The samples were processed into small pieces and grinded in a mortar and pestle with a small amount of distilled water. Approximately 10 g of grinded mixture was taken into screw capped test tube. About 15 ml of distilled water, 0.5 ml of chloroform and 1 ml of concentrated sulphuric acid were added into the test tube. The picrate paper was kept into the mouth of the test tube and closed with the screw cap. Test tubes were kept in the boiling water bath until the samples in the tube starts boiling. Test tubes were allowed to cool and observed for the picrate paper for interpretation.

RESULTS AND DISCUSSIONS

The level of HCN required to cause toxicity varies, depending on rate of intake and individual animal tolerance. Generally speaking, view as dangerous any forage or leaves analyzing more than 200 ppm HCN on an as-fed basis. The clinical signs of the toxicity observed during the investigation were salivation, lacrimation, dyspnoea, laboured and quick breathing, and bloat followed by death. Death occurred in six Malnad Gidda cattle within a span of 24-48 h. The clinical signs and mortality were in accordance with[3]. Post-mortem findings showed bright red coloured blood, congested liver and intestine, and distended rumen with gas[4]. The clinical signs and mortality observed in cattle were due to a presence of toxic principle linamarin, which is a Cyanogenetic glycoside[5] in *Hevea brasiliensis* leaves. HCN is one of the most potent, rapid-acting poisons known. At cellular level cyanide radical combines with the ferric iron of cytochrome oxidase enzyme and inhibits the electron transport system in mitochondria and hence inhibits cellular respiration[6]. When a lethal dose is consumed, animals die from asphyxiation in a few minutes[7]. The young rubber tree leaves which were analysed for the detection of HCN, a toxic principle by Picric acid paper spot test, showed positive reaction. The Picric acid paper turned from yellowish to brownish brick red colour (Figure 2). The change in colour on the picrate paper could be attributed to release of HCN from leaves[6]. The linamarin, a cyanogenetic glycoside present in *Hevea brasiliensis* leaves, releases HCN gas which reacts with picric acid to give colour change. Whereas Post-mortem samples showed

no change in the colour, suggestive of absence of HCN which may be due to rapid absorption and escape of HCN from the rumen into circulation and redistribution to different parts of the body tissues. HCN causes acute poisoning in ruminants grazing young rubber tree leaves. Many of the same factors that tend to cause nitrate accumulation – drought, reduced sunlight, excessive soil nitrogen, and young plants, also increase HCN potential. HCN potential is greater in leaves than stems. Proper curing for rubber tree leaves or ensiling greatly reduces the potential for HCN poisoning. Treatment of HCN poisoning, with a mixture of sodium thiosulfate with methylene blue, can be successful if administered by a veterinarian soon after symptoms appear[7].

Figure 1: *Hevea brasiliensis* leaves

Figure 2: Picric acid paper spot test Negative control and Positive *Hevea brasiliensis* leaves

Summary

The present study was undertaken to identify the aetiology of sudden mortality in Malnad Gidda cattle. The clinical observations in the ailing animals and post-mortem findings in the carcasses revealed toxic effects of *Hevea brasiliensis* immature leaves. The Picric acid paper spot test confirmed the presence of cyanogenetic glycoside in the leaves. The present study revealed the toxic effects of *Hevea brasiliensis* in Malnada Gidda Cattle grazed on rubber tree accidently. The poisoning due to *Hevea brasiliensis* can be reduced with proper curing for leaves or ensiling before fed to animals.

CONCLUSION

In the present study, deaths occurred in six Malnad Gidda cattle within a span of 24-48 h. The clinical signs of the toxicity observed during the investigation were salivation, lacrimation, dyspnoea, laboured and quick breathing, and bloat followed by death. The Picric acid paper spot test of the *Hevea brasiliensis,* rubber tree leaves revealed the presence of cyanogenetic glycoside.

REFERENCES

1. Norman R. Overview of Cyanide Poisoning. The Merck Veterinary Manual; 2012.
2. Flanagan RJ, Braithwaite RA, Brown SS. Basic Analytical Toxicology. Geneva: WHO; 1995.
3. Cran HR. Suspected hydrocyanic acid poisoning in cattle. Veterinary record 1985; 116: 349. http://dx.doi.org/10.1136/vr.116.13.349
4. Burrows GE, Way JL. Cyanide intoxication in sheep: Antagonism with sodium nitrite, cobalt chloride and sodium thiosulfate. American Journal of Veterinary Research 1979; 40: 613.
5. List PH, Horhammer L. Hager's handbuch der pharmazeutischen praxis. Berlin: Springer; 1979.
6. Harpal SS, Rajinder SB. Textbook of Veterinary Toxicology: Plants containing Cyanide. 2nd ed. Ludhiana: Kalyani Publishers; 2009. p. 281-288.
7. Whittier JC. Prussic Acid Poisoning. US: Colorado State University; 2014.

Cite this article as:

Lohith T.S, Venkatesha M.D, Mallinath K.C, Sobharani M, Shankar B.P. *Hevea brasiliensis* poisoning in Malnad Gidda Cattle, Karnataka, India. Int. Res. J. Pharm. 2014; 5(7):578-579 http://dx.doi.org/10.7897/2230-8407.0507117

Source of support: Nil, Conflict of interest: None Declared