

Sorghum Vulgare (Jowar) Poisoning in Sheep

D. C. Shukla, J. V. Anjaria and D. D. Heranjal

Gujarat College of Veterinary Science and

Animal Husbandry, GAU, Anand Campus, Anand-388 001

ABSTRACT

Sorghum vulgare (Jowar) poisoning due to grazing of its ratoon crop is reported in seven sheep, three of which were saved by intravenous administration of sodium nitrate (2%) and sodium thiosulphate (5%) in 50 ml dose. Acute toxic symptoms aided by autopsy findings and sodium picrate paper test indicated poisoning by hydrocyanic acid; which was isolated from ratoon crop of *Sorghum vulgare* consumed by the sheep and also from the ruminal contents collected at autopsy.

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INTRODUCTION

Reports on the *Sorghum vulgare* (Jowar) poisoning in animals in India are few. Maji (1930) reported jowar poisoning in six cows. Bagchi and Ganguly (1941) stated that poisoning of *Sorghum vulgare* was common in cattle of Bengal and Bihar. It is the common experience of the farmers and shephards that very young plants of jowar are very toxic to the livestock. *Sorghum vulgare* plants contain cyanogenic glucoside which liberates hydrocyanic acid, which when absorbed leads to fatal poisoning in all species of animals and particularly ruminants are more susceptible (Holmes 1966). *Sorghum vulgare* and some other cyanogenic plants used as forage may also cause heavy mortality when fed at a stage with high cyanogenic glucocide content. The poisoning is

caused by ingestion of cyanogenic plants that are capable of producing hydrocyanic acid (Sippel 1970). The hydrocyanic acid (HCN) content is more when plants grow rapidly after a previous period of retarded growth (Blood and Handerson 1974). and HCN concentration is more in the stem than in the root and leaves when they grow 3 to 5 inches long (Bagchi & Ganguly, 1941). It is observed that HCN concentration is very heavy in the ratoon crop (Re-sprouted plant after cutting) of *Sorghum vulgare*. In the present paper, a sporadic incidence of *Sorghum vulgare* poisoning in sheep has been reported.

MATERIALS AND METHODS

Seven sheep belonging to the Gujarat Veterinary College Instruction Farm were reported to have grazed on

rutoon crop of *S. vulgare* in October 1978. All the animals started showing symptoms of poisoning after about half an hour of grazing. The symptoms were characterised by dyspnoea, restlessness, moaning, recumbancy, terminal clonic convulsions, opisthotonus and bright red mucous membrane. Four animals out of seven died within an hour. The rest three showed less acute symptoms of hyperaesthesia, dilatation of pupils and nystagmus. They were immediately treated with the following freshly prepared solution intravenously. Sodium nitrate, 1 g., sodium thiosulphate, 2.5 g and distilled water 50 ml. The dead sheep were subjected to autopsy. Tissues were collected from different organs in 10% formalin for histopathological investigation. They were processed and embedded in paraffin blocks. Sections of the blocks were cut to 5-6 μ thick and stained with hematoxyline and eosin stain for microscopic examination.

Sodium picrate paper spot test was carried out both on the ruminal contents of the freshly dead sheep and on the stunted plants of *S. vulgaris* as well as its rutoon crop which the sheep had consumed prior to death. Strips of filter paper dipped in 10% sodium bicarbonate and 1% picric acid solution were kept dried. The strips were kept hung in the glass stoppered bottles containing the test material treated with chloroform. The bottles were kept in the incubator at 37° C.

RESULTS AND DISCUSSION

The picrate paper spot test was found positive for HCN poisoning as

indicated by the appearance of the pink coloration of the picrate paper on incubation of the test material. All the sheep treated with sodium nitrite and sodium thiosulphate solution recovered completely within six hours. The intravenous administration of the solution appears to have prevented the toxic activity of the highly lethal cyanide ions in the body tissues by their conversion into thiocyanide and consequent excretion of the same through the kidneys. Autopsy of the dead sheep revealed no significant gross lesions. However, the blood of the freshly dead carcasses was found to be bright red in colour which is suggestive of HCN poisoning also. Histopathological lesions were characterised by fatty changes of the hepatic cells, extensive haemorrhages accompanied by hemosideroids in the spleen, early degenerative changes in the renal tubular epithelium and lymphoid hyperplasia of the lymph nodes. These lesions were indicative of the effects of the toxicity of the parenchymatous organs by the HCN.

Jowar crop which when remains unwatered for a long time during the period of its growth, contains high concentration of cyanogenic principles. The nonwatered rutoon crop of *Sorghum vulgare* were observed to be having a very high concentration of cyanogenic principles. The cases reported in the present paper were so acute that the death was quick. The glucosides when hydrolysed in the rumen by enzymes of the ruminal microflora, produce HCN which inhibits the

activity of the cell enzyme, cytochrome oxidase resulting in disturbed cell respiration and subsequent cellular death including the vascular endothelium that caused haemorrhages in the parenchymatous organs. Great care is therefore necessary for keeping the animals away from consuming stunted plants of jowar to prevent HCN poisoning. Our findings reveal that in case the animals show the symptoms of HCN poisoning, they must be immediately treated with freshly prepared sodium nitrite and sodium thiosulphate solution for early recovery and animals should be prevented from further grazing on such crop.

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