Management of Hydrocyanic Acid Intoxication in a Flock of Goats due to Weed Grazing after Rain in a Drought Climate

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Abstract

A flock of thirty seven goats following grazing suddenly developed signs of tympany, salivation, respiratory distress, dyspnoea, gasping and prostration. All the goats were administered with sodium thiosulphate 660mg/kg B.wt. intravenously and the goats recovered uneventfully. The occurrence of HCN toxicity and its emergency management after ingestion of unknown weed plantsis reported in this paper.

Key words: *HCN poisoning, goats, weed plants.*

Cyanogenic plant toxicity is one of the most common plants poisoning among the grazing livestock. Ruminants are more susceptible to poisoning by cyanogenic plants, than horses and pigs due to more efficient hydrolysis of the cyanogenic glycosides in ruminants *(Clarke,* 1981). Hydrocyanic acid poisoning are caused by the ingestion of plants containing cyaonogenic glycosides like sugarcane top sorghum, sudan grass etc. The glycoside is non-toxic, which leads toits toxicity to cyanide after hydrolysis. Within the group of ruminants, goats appear to be the most susceptible to cyanide. (*Patel,* 2012).This paper records an unusual case of HCN intoxication due to grazing of weed plants in drought climatic condition after a short rainfall.

Case History and Observations

A flock of about fifty goats were let for grazing by the owner from the indoor rearing after a season of rain in a drought area. In the evening, out of fifty goats, thirty seven goats suddenly developed bloat, staggering gait, in coordination, salivation and dyspnoea. Death was observed immediately in five goats. All the animals were immediately shifted to the Teaching veterinary Clinical Complex, VCRI, Namakkal for treatment. Clinical examination revealed bloat, ptyalism, dyspnoea, oral breathing, gasping, dilated pupil, tachycardia and brick red colour conjunctival mucous membrane.

Treatment and Discussion

Based on the clinical signs, HCN intoxication was suspected and all the animals were administered with intravenous injection of sodium thiosulphate @ 660 mg/kg B.wt. immediately after administration of the sodium thiosulphate. All the animals started urination and reduction in severity of clinical signs. After one hour of treatment. All the animals recovered uneventfully and became active and alert. Incidences of cyanide intoxication were reported in goats in California after eating clippings from California Holly

(Heteromelesarbutifolia) which contains cyanogenic glycosides. Within four hours of feeding, three animals died, and seven were moribund (*Tegzes J.H.*, 2003). 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 enzyme. Hydrogen cyanide is absorbed readily from the gastrointestinal tract and enters individual cells. Acute intoxication causes tissue anoxia by inhibiting cytochrome oxidase and prevents cellular respiration. The earliest signs after cyanide exposure are due to dysfunction in neurons and in the myocardium and are manifested most commonly as collapse. Hypotension and tachycardia may be observed. Further damage to neurons arises secondary to systemic hypoxia and ischemia. (*Tegzes et al, 2003*)

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