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Successful management of hydrocyanic acid poisoning in a doe

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Abstract
A three-year-old, non-descript doe was presented with a history of acute onset of respiratory distress, distended abdomen, salivation and muscle tremor. The animal was allowed freely to graze in an immature fodder sorghum field, which was sown two weeks ago and poorly irrigated. Clinical examination revealed bright red mucus membrane and vertical nystagmus. Based on these, hydrocyanic acid poisoning was diagnosed and was treated with intravenous sodium nitrite followed by sodium thiosulphate and supportive therapy. This treatment resulted in excellent resolution of clinical signs and an uneventful recovery of the animal.

Keywords: Goat, HCN, hydrocyanic acid, sodium nitrite, sodium thiosulphate

Introduction
Hydrocyanic acid (HCN) or cyanide poisoning is one of the most common poisonings in ruminants that are reared in semi-intensive and extensive farming methods. The major source of cyanide is under irrigated immature sorghum fodder. These cyanogenic plants contain cyanogenic glycoside (> 750ppm) instead of free cyanide and are potentially hazardous to animals. Elevated environmental temperature and the use of nitrogenous fertilizers result in increased cyanogenic glycosides in plants (Karthika and Kalpana, 2017) [4]. Mastication and salivation of those plant molecules release the cyanide from cyanogenic glycosides. Ruminants are more prone to develop HCN poisoning compared to monogastric animals due to the high release of cyanide components from the plant molecules by the action of the ruminal microbiota. Ingestion of a large quantity of cyanogenic plants causes 100% mortality (Gensa, 2019) [3]. This case report describes the diagnosis and successful management of hydrocyanic acid poisoning in a doe.

Case Description
A three-year-old, non-descript doe weighing 28 kgs was presented with a history of acute onset of respiratory distress, distended abdomen, salivation (Fig.1) and generalized shivering. The animal was allowed freely to graze in an immature fodder sorghum field, which was sown two weeks ago and poorly irrigated. One more animal from the same flock died with similar clinical signs on the same day as per the owner's report.
Clinical examination revealed bright red mucus membrane (Fig.2), nystagmus, muscle tremor, bloated abdomen, tachycardia (120 beats /min) and Tachypnea (38 breaths/min).
Based on these, hydrocyanic acid poisoning was diagnosed tentatively. The treatment was initiated with Sodium nitrite at the dose of 20 mg/kg diluted with 100 ml of normal saline followed by Sodium thiosulphate (Fig.3) at the dose rate of 660 mg/kg diluted with 100 ml of dextrose normal saline, I/V, Inj. Vitamin B1, B6, B12 (Tribivet) 2ml I/V and Inj. Chlorpheniramine maleate 2ml I/M as total dose. In same time, rumen fluid was collected by needle aspiration and subjected to a picric acid paper test (Qualitative test). The appearance of yellow-brown color in the filter paper (Fig.5) indicated the presence of hydrocyanic acid in the rumen sample and by which this case was confirmed as hydrocyanic acid poisoning.
The animal showed complete resolution of clinical signs and uneventful recovery on subsequent days (Fig.4).

Discussion
Plants containing a toxic quantity of cyanoglycosides are responsible for hydrocyanic acid poisoning in ruminants.

The common cyanogenic plants were sorghum (dhurrin), bitter almond (amygdalin) and tapioca root (linamarin) (Burrows and Tyrl, 2013) [2]. Rumen microflora rapidly breakdowns plant molecules and releases the free cyanine from cyanoglycosides. This cyanideis readily absorbed and enters into the systemic circulation and results in the formation of cyanomethemoglobin (Arnold *et al.*, 2014) [1]. The brain and heart are the organs affected at the earliest due to significantly reduced oxygen-carrying capacity by methaemoglobin and exhibit clinical signs.

The common clinical signs associated with acute cyanide poisoning in ruminants are tachypnea, dyspnea, tachycardia, bloat abdomen, nystagmus, bright cherry red mucus membrane, bitter almond breath smell, muscle fasciculation, coma and death. Clinical signs start within 15-20 minutes to a few hours after consuming the cyanogenic plants (Muwel *et al.*, 2018). The main cause of death due to acute cyanide poisoning is histotoxic anoxia. The lethal dosage of HCN in most animal species is 2 -2.5 mg/kg.

Immediate treatment includes hydroxocobalamin (B12) (70 mg/kg) and oxygen therapy. Rapid IV infusion of sodium nitrite at the dose rate of 20 mg/kg over 3-5 mins followed by sodium thiosulphate at the dose of 660 mg/kg is recommended (Rajasokkappan *et al.*, 2020) [6]. Treatment with intravenous sodium thiosulphate alone has shown successful results and should not be delayed for diagnostic confirmation.

Fig 1: Distended abdomen and salivation

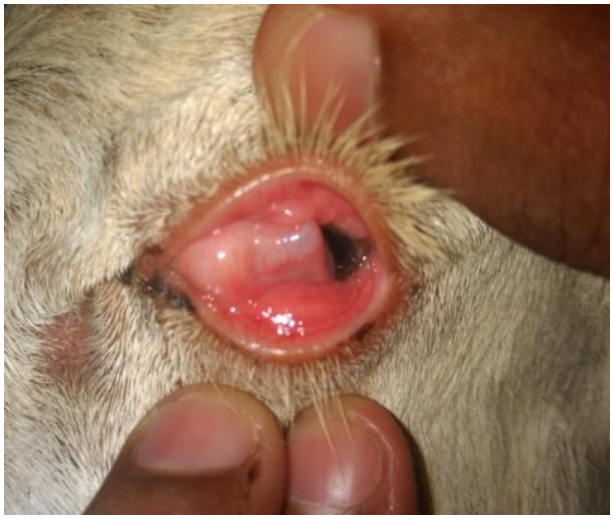

Fig 2: Bright red mucus membrane

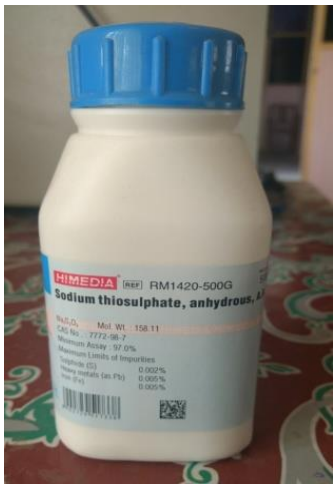

Fig 3: Sodium thiosulphate

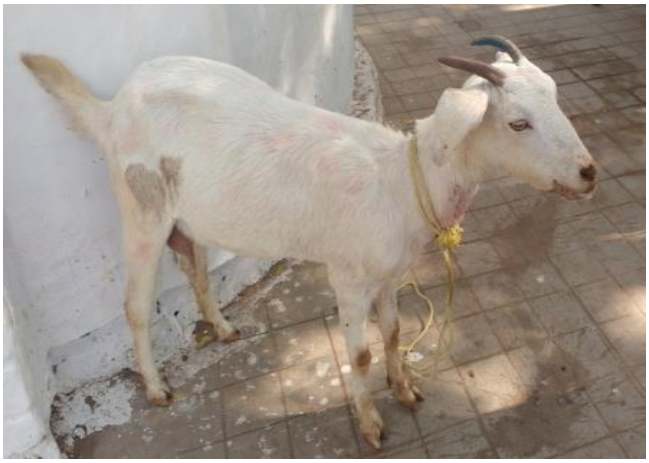

Fig 4: After treatment

Fig 5: Picric acid paper test

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