



# Cyanide Poisoning in Animals and Its Management

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## Abstract

Cyanide words refer as a toxin to ruminants such as cattle, sheep, goats. This toxicity occurs when animals graze the plant which contain cyanogenic glycosides. Cyanide contains plants are Choke-cherry, *Sorghum sudanense*, Johnsongrass, Laurel. Symptoms can develop within minutes to hours after ingestions of cyanide contain plants. Treatment involves antidotes, sodium thiosulphate and sodium nitrate, oxygen therapy. Prevention mainly focused on avoiding the plants which considers as cyanide toxicity.

**Keywords:** Cyanide poisoning, toxicity, ruminants, symptoms, prevention.

## Introduction

Cyanide is considered as a poison to animals which have the cyano group (C-N) in their structure and it is highly toxic. Hydrocyanic acid as well as hydrogen cyanide are acidic in nature and referred as harmful. Gaseous HCN, water-soluble potassium and sodium cyanide, and weakly water-soluble mercury, copper, gold, and silver cyanide salts are also other forms of cyanide (Enah, 2024).

Cyanide exposure in animals occurs more often when they consume plants that contain cyanogenic glycosides. Cyanogenic glycosides, which are chemicals that produce cyanide, are the main source of cyanide poisoning in ruminants. There are at least 55 known cyanogenic glycosides found in plants, many of which are produced during regular plant metabolism from amino acids. But among cyanogenic plants, the most prevalent glycosides have been identified as Amygdalin, Prunacin, Linamarin, Lotaustralin,

Dhurryn, Taxiphylyn, Vicianyn, Proteacynve, and Gynocardyn (Gensa, 2019).

In India, cyanogenic plant poisoning is frequently caused by Choke-cherry, wild cherry, elderberry, and arrow grass. Other common causes include giving livestock immature sorghum, such as young shoots of *Sorghum vulgare* and *Sorghum sudanense*, Johnson grass and other forage sorghums like grain sorghum, sheep and goats inadvertently consuming *Acacia leucocephala* pods (Bates, 2022).

One of the toxins that affects cattle the fastest is cyanide; poisoning usually happens after cyanogenic plants are consumed.

Most harmful cyanogenic plant is *Prunus laurocerasus* and more than thousand species of cyanogenic plants contains cyanogenic glycoside chemical. In the plant cells, enzymes are responsible for conversion of cyanogenic glycoside into the free cyanide gas. When the

plant cells are destroyed by chewing, crushing or drooping the plant cell enzymes combine with cyanogenic glycosides and produce hydrogen cyanide (Kennedy *et al.*, 2021).

### How animals affect by cyanide poisoning

Ruminants have enzyme in their rumen microflora which rapidly break the cyanogenic glycosides and it make more prone to cyanide poisoning than in monogastric animals. The rumen pH is high and bloodstream absorbs HCN as and when it is released in rumen. The affected animal suffering from anoxia and this prevents the mitochondrial electron transport. Cattle and sheep can metabolize less quantity of cyanide to thiocyanate in the liver and eliminate through urine. Cyanide is extremely powerful, though, and when large amounts are taken quickly, the body's detoxification systems are overloaded, frequently leading to death in less than two hours (Agustin *et al.*, 2022)

### Pathogenesis

By paralyzing tissue enzymes, acute cyanide intoxication results in histotoxic anoxia and subsequent tissue hypoxia. Dyspnea is the main symptom for cyanide poison due to anoxia. Convulsions, cerebral anoxia, muscle tremor, blood giving distinctive brilliant red color develop due to anoxia. Further cyanogenetic glycosides can lead to bloat in ruminants (Dalefield, 2017).

Cyanide rapidly enters the central nervous system (CNS) during acute exposure, inhibiting oxidative phosphorylation and disrupting aerobic metabolism, which causes immediate neurological damage. The brain's inadequate energy stores and restricted anaerobic ability cause this severe cyanide sensitivity. On the other hand, long-term exposure to cyanide may have distinct effects on the central nervous system, possibly leading to the degeneration of particular brain regions (de Sousa *et al.*, 2025)

**Clinical Signs and Symptoms** (Sankaran *et al.*, 2020)

**Acute poisoning from cyanide:** - Animals rarely survive more than two hours after consuming poisonous forage, and symptoms typically appear 15–20 minutes to a few hours later.

At first, there may be signs of excitement and a fast-breathing rate. Soon after, there is tachycardia and dyspnea. There may be the traditional "bitter almond" breath odor.

There may be excessive lacrimation, voiding of urine and feces, and salivation in pigs.

Muscle fasciculation can cause spasms and comatose condition develop before death. Animals suffering from struggle and stumble before collapsing. At first the mucous membranes are bright red color and at the end turn into cyanotic. Because of high amount of dissolved Oxygen in venous blood it becomes "cherry red".

Increased level of serum ammonia, neutral and aromatic amino acids are observed. Myocardial histotoxic hypoxia causes cardiac arrhythmias and the cause of death is asphyxial convulsions.

**Chronic cyanide poisoning:** - Cyanogenic glycosides leads to chronic hypothyroidism with or without goiter. Incoordination can lead to non reversible flaccid paralysis, and urinary incontinence is related to cystitis. Pyelonephritis is frequently linked to death but it is not common. Musculoskeletal teratogenesis and late-term abortion are also possible outcomes.

**Treatment** (Giantin *et al.*, 2024; Sinha *et al.*, 2019)

- Oxygen should be given it is available, particularly for small animals.
- The most effective counter agent or antidotes for cyanide poisoning is Hydroxocobalamin (vitamin B12a) @ 70 mg/kg administered intravenously over 15 minutes is the recommended dosage, which can be repeated as needed.
- Standard treatment involves intravenous administration of a combination of sodium thiosulphate and sodium nitrate at 66 and 22 mg/kg weight.
- 5% Dextrose administration IV to dilute the toxin and as a supportive therapy.
- Injection Anistamin (Chlorpheniramine maleate 10 mg/ml) IM and Curadex (Dexamethasone Sodium 4 mg/ml) ml IM.
- Rumenotomy can be conducted to transfer normal rumen content. The affected rumen contents should be withdrawn and replaced with those from a healthy animal.

**Prevention for cyanide poisoning** (Arnold *et al.*, 2014; Deen *et al.*, 2018)

- The following management techniques can help lower the risk from potentially hazardous forages: - Because cyanogenic glycoside

- concentrations are highest in young, fast-growing plants or regrowth, especially in the newest leaves and sensitive tips, graze johnson grass, sorghum crosses, or sorghum when they are at least 18 to 24 inches tall. It is not appropriate to graze immature plants. Wait 4 to 5 days after rainfall after a drought before grazing.
- b) Avoid grazing plants when there is a drought because this will cause the plant to wilt or twist or its growth to be drastically inhibited. Due to delayed growth and the plant's inability to mature, drought increases the likelihood of cyanide by encouraging the production of cyanogenic compounds in the leaves.
  - c) When there is a chance of frost, especially at night, avoid grazing potentially dangerous forages. Within the plant, frost permits conversion to hydrogen cyanide. Avoid grazing sorghum for 2 weeks after a non-killing (>28 degrees) frost.
  - d) Avoid feeding green chop if there is a suspicion of excessive cyanide in the forages. Allow the hay to completely dry before baling to allow the cyanide to volatilize. Because toxicity might be retained in cool or damp temperatures, allow for slow and complete drying.
  - e) Six to eight weeks after ensiling, postpone feeding silage. Low cyanide potential can be used to choose forage species and types. Plant varieties differ greatly from one another. Piper is one of the Sudan grasses that has a low cyanide content.
  - f) Examine any dubious forages before giving animals access to them. On-site findings can be obtained with a quick field test. For further information, speak with the country Agricultural Extension representative.

## Conclusion

Cyanide toxicity is one serious poisoning in animals. Ruminants can be easily affected by this toxin because of their feeding habits. Death may occur in affected animal. The poison can be neutralized if the treatment is administered quickly, although the toxin's fast-acting nature usually results in the animals' deaths. It is important to focus on preventing this toxication.

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