# Cyanide Poisoning

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Sources/Causes:

1. Fumigants, soil sterilizers, fertilizers, pesticides/rodenticides
2. Component of internal combustion engine exhaust and tobacco smoke.
3. In livestock species, the most frequent cause of acute and chronic cyanide poisoning is ingestion of plants that either contain cyanogenic glycosides or are induced to produce cyanogenic glycosides and cyano-lipids
4. Cherry, peach, plum and bitter almond also includes cyanide. Sorghum spp (Johnson grass, Sudan grass the common cereal grain crop referred to as "sorghum" or the synonyms durra, jowar, milo), Acacia, linseeds and flaxes, marsh arrow grasses, cassava), all members of the Prunus genus. Plant materials containing ≥200 ppm of cyanogenic glycosides are dangerous.

Mechanism of action

1. Cyanide taken to the body
2. it's rapidly absorbed and circulated
3. It is merged with methemoglobin and forms cyanomethemoglobin.
4. The circulating cyanide inactivates cytochrome oxidase enzyme by binding ferric (Fe+++) iron which is within this enzyme.
5. Normally the cytochrome oxidase enzyme catalyzes the last step of oxidative phosphorylation.
6. The enzyme-cyanide complex prevents this task from being performed.
7. The enzyme cannot combine with oxygen and electron transportation become inhibited.
8. The animal cannot use oxygen and cellular respiration stops immediately.
9. Death occurs due to histotoxic anoxia.

Clinical signs:

1. Cyanide can cause death in a short amount of time.
2. The toxicity depends on amount of ingested cyanogenic structure and ingestive rate.
3. Affected cattles rapidly begin to show toxication in 5-15 minutes to a few hours, but usually do not survive 2 hours after consuming the lethal dose of cyanogenic structures.
4. Dyspnea, labored breathing, restlessness, tremors, groaning, terminal clonic convulsions and opistothonus are the clinical signs in affected animals.

5. Initially bright and cherry-red colored mucous membranes are noticed.
6. When patient becomes hypoxic, mucous membranes become cyanotic
7. Symptoms of poisoning occurs due to hypoxia .
8. In chronic poisonings, arthrogryposis can be observed in the calves which consume sorghum- Myelomalacia and urinary incontinence, Incoordination, ataxia, head shaking and pawning in the posterior extremities.
9. Decrease of productivity, difficulty on conceive and abortion can be also seen .

Diagnosis

1. Appropriate history, clinical signs, post-mortem findings, and demonstration of HCN in rumen (stomach) contents or other diagnostic specimens support a diagnosis of cyanide poisoning.
2. Cyanide analysis must be performed on suspected feeds and plants or cattle's rumen content for the diagnosis
3. Nonspecific early symptoms of toxicity such as dizziness, weakness, diaphoresis, hyperpnea and labored breathing. collection of examples is very important.
4. In necropsy, early formation of death attendance can be observed. Blood can be seen in red colour Red and orange colour in the mouth and stomach can be observed.
5. Bitter almond odor can be smelt in rumen and internal organs. Subendocardial and sub-pericardial hemorrhages occur in all cases .
6. Differential diagnosis- acute cyanide poisoning can be misdiagnosed with nitrate poisoning, organophosphorus poisoning and sulphur poisoning.

Treatment

1. sodium nitrate and sodium thiosulfate should be rapidly administered intravenously to affected cattles.
2. Rumen content should be removed and replaced with contents from an healthy animal
3. Sodium nitrite and sodium thiosulphate are the specific antidotes for cyanide poisoning 20% of sodium nitrate and 20% of sodium thiosulphate mixture can be applied intravenously to animals.
4. After the treatment the respiratory rate become slower and the cattle seemed to be more relaxed.
5. Animals must be placed in a position of sternal recumbency and an attempt must be done to rise immediately.

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