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Popular Article

Postmortem Changes, diagnosis and treatment of Cyanide toxicity in animals

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Introduction

Exposure to cyanide, a very toxic substance present in many natural and industrial sources, can result in cyanide poisoning, a disease that can be fatal. Ruminant animals (cattle and sheep) are more prone to cyanide poisoning than nonruminant animals because the ruminal microorganisms have enzymes that will release cyanide in the animal's digestive tract.

Etiology

The primary cause of cyanide poisoning in ruminants is the ingestion of plants containing cyanide-producing compounds called cyanogenic glycosides. The most common plants that cause cyanide poisoning are Sudan grass and sorghum. Other plants, however, have the potential to produce cyanide. Plants with high amounts of cyanide: Apple, Apricot, Arrow Grass, Cherry, Chokecherry, Flax, Forage sorghums, Grain sorghums with toxic glycosides like amygdalin.

Factors affecting cyanide toxicity

- Leaves usually produce two to 25 times more cyanide than do stems in forage grasses; seeds contain no cyanide. Young, upper leaves have more cyanide than lower leaves.
- The amount of cyanide in plants which are cultivated in soils that high in nitrogen and low in phosphorus seems to be higher.
- Herbicides like 2, 4-D have the ability to raise cyanide levels in forage for several weeks following application. Plan grazing rotations accordingly.

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- High cyanide levels in plants are more likely to occur during drought. This could be as a result of the plants have not been able to mature and the fact that they are primarily made up of cyanide-rich leaves. In general, higher than normal amounts of cyanide can result from any stress environment that slows down plant growth.
- Frost or freezing causes plant cells to rupture, allowing cyanide to be released.
- The rumen microbiota also contains beta-glucosidase and hydroxynitrile lyase, and a pH of 6.5 to 7.0 in the rumen promotes the conversion of cyanogenic glycosides to cyanide.

Mammals use mitochondrial rhodanese to convert about 80% of the cyanide they consume into thiocyanate under low-level exposure situations. Thiocyanate is then largely excreted in urine. The availability of thiosulfate is a common factor that restricts the pace of the rhodanese pathway. It's also important to note that dogs have lower total rhodanese activity than other species.

The nonenzymatic cyanide-cysteine reaction produces beta-thiocyanoalanine, which is then transformed into 2-iminothiazolidine-4-carboxylic acid and excreted by mammals. Cyanide can also be detoxified by combining with hydroxycobalamin (vitamin B12a) to produce cyanocobalamin (vitamin B12). Low dietary intakes of sulfur aminoacids (L-cysteine and L-methionine) will result in higher blood cyanide levels, particularly under conditions of chronic, low level exposure. It is well established that dietary sulphur and sulfur-containing amino acids have a significant impact on the neurologic toxidromes linked to prolonged exposure to cyanide and cyanogenic glycosides.

Pathogenesis

Cyanide ion reacts with Fe^{+3} (ferric) ion of cytochrome oxidase to form a stable complex

↓

Conversion of Fe^{+3} to Fe^{+2} is thereby prevented so that electron transport and cellular respiration are halted. The blood is oxygenated, but cannot be utilized by the cells.

↓

lack of O_2 utilization in chemoreceptors and/or neurons of the brain triggers increased respiratory efforts and the blood becomes hyper oxygenated (bright red). End result is histotoxic anoxia.

Lactic Acidosis

When cellular respiration is compromised, cells shift to anaerobic metabolism, which produces lactic acid as a byproduct. This condition is known as lactic acidosis. The body's acid-base balance is made worse by lactic acidosis, which results from an accumulation of lactic acid in the blood.

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Multi-Organ Failure

The combined effects of cellular hypoxia, acidosis, and cardiovascular collapse can lead to multi-organ failure, affecting vital organs like the heart, liver, kidneys, and lungs.

Clinical Signs

Acute cyanide poisoning

Signs generally occur within 15–20 minutes to a few hours after animals consume toxic forage and survival after onset of clinical signs is rarely >2 hours. Excitement can be displayed initially, accompanied by rapid respiration rate. Dyspnea follows shortly with tachycardia.

There may be a characteristic "bitter almond" breath odor, excessive lacrimation and voiding of urine and faeces may also occur. Vomitions may occur, especially in pigs. Muscle fasciculation is common and progresses to generalized spasms and coma before death.

Animals may stagger and struggle before collapse. In other cases, sudden unexpected death may ensue. Mucous membranes are bright red but may become cyanotic terminally. Venous blood is classically described as "cherry red" because of the presence of high venous blood pO_2. However, this color rapidly changes after death. There is often an increase in serum ammonia as well as neutral and aromatic amino acids. Myocardial histotoxic hypoxia is a prevalent cause of cardiac arrhythmias.

Severe asphyxia convulsions result in death. After struggling, the breathing ceases but the heart may still beat for a few minutes. The prognosis for recovery without therapeutic intervention is poor because as the elimination half-life of cyanide in dogs is known to be 19 hours, it would take more than 4 days to eliminate >95% of the cyanide present.

Chronic cyanide poisoning

It manifests as chronic cyanogenic glycoside hypothyroidism, with or without goitre. Cystitis ataxia toxidromes are typically associated with posterior ataxia or incoordination that may progress to irreversible flaccid paralysis, cystitis secondary to urinary incontinence and alopecia. Despite being rare, death is frequently linked to pyelonephritis. There may also be musculoskeletal teratogenesis and late-term abortion.

In horses and ruminants, neuropathy syndromes are linked to chronic, low-level cyanide/cyanogenic glycoside exposure (frequently in conjunction with insufficient dietary sulphur and/or Sulphur amino acid intake). The lateral and ventral funiculi of the spinal cord and brain stem exhibit widespread nerve fibre degeneration in Sorghum cystitis ataxia syndrome in horses. Similar syndromes have been described in ruminants.

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Postmortem changes

Understanding the postmortem changes associated with cyanide poisoning can be crucial in determining the cause of death.

Livor Mortis

The Cherry-Red Clue One of the most striking postmortem changes in cyanide toxicity is the presence of cherry-red discoloration in the dependent areas of the body, a phenomenon known as livor mortis.

Mucous membranes may also be pink initially, then become cyanotic after respiration ceases Congestion and oedema: Cyanide toxicity can lead to congestion (accumulation of blood) and oedema (fluid buildup) in various organs, including the brain, lungs, and liver.

- Acute haemorrhages: Haemorrhages (bleeding) may occur in a variety of organs, most notably the brain.
- Brain changes: The brain may appear congested, swollen, and may exhibit petechial haemorrhages (small pinpoint haemorrhages).
- Respiratory changes: Pulmonary oedema (fluid accumulation) and congestion may be visible in the lungs.
- Changes in the liver and kidneys: The liver and kidneys may show signs of haemorrhage and congestion.
- Generalized dark coloration of skeletal muscle

Histopathological findings

A histological examination (a microscopic examination of tissues) may reveal histological abnormalities, such as cell necrosis and degeneration, that are typical of cyanide poisoning, especially in the brain and heart.

Diagnosis

- History of consumption of cyanogenic plants or cyanide salts
- Analysis of forage, blood, rumen contents, liver, muscle
- Analysis of blood for cyano-haemoglobin
- Sodium picrate paper test.

Differential diagnoses include poisonings by: nitrate or nitrite, urea, organophosphates, carbamates, chlorinated hydrocarbon pesticides, toxic gases (carbon monoxide and hydrogen sulfide) as well as infectious or noninfectious diseases and other toxidromes that cause sudden death.

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Treatment

- Sodium nitrite is injected intravenously to convert hemoglobin to methemoglobin, which reacts with cyanide from the cyanide-cytochrome complex to form cyanmethemoglobin. A simultaneous injection of sodium thiosulfate provides sulfur to convert cyanmethemoglogin to the less toxic thiocyanate, which is excreted in the urine. The remaining methemoglobin is converted by other enzymes to hemoglobin, which then is available to transport oxygen normally.
- Immediate treatment with hydroxocobalamin and oxygen
- Methylene blue if diagnosis is in doubt (signs are similar to those of nitrate poisoning)
- Removal from the source of exposure

Caution

Clinical signs of cyanide poisoning and nitrate poisoning are quite similar. Be certain nitrates are not a problem before administering sodium nitrite. An injection of sodium nitrite into an animal already suffering from nitrate poisoning would be disastrous.

Sodium thiosulfate, alone, is also an effective antidotal therapy for cyanide poisoning.

Prevention

- Never graze sorghum less than 18 inches in height.
- Feed hungry cattle before allowing them to graze forages that may contain high levels of cyanide.
- Do not allow animals to graze troublesome plants after a light frost or after rain has ended a summer drought. Wait several days after a killing frost before grazing.
- Chop or ensile plants high in cyanide to reduce toxin levels.
- Analyze suspect forage samples before feeding.

Conclusion

Postmortem changes in cyanide toxicity add a layer of intrigue to forensic investigations, guiding experts in understanding the circumstances surrounding an animal's tragic death.

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